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VOLUME XXXVIII FEBRUARY, 1924 NUMBER 2

Surgery, Gynecology and Obstetrics

WITH

International Abstract of Surgery

Official Journal of the American College of Surgeons

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PUBLISHERS FOR GREAT BRITAIN: Bailliere, Tindall & Cox, 8 Henrietta St., Covent Garden, London, W. C., England

Published Monthly. Annual Subscription: United States and Canada, $12.00; Foreign, $14.00.
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Entered as second-class matter May 22, 1913, at the postoffice at Chicago, Ill., under the Act of Congress, March 3, 1879.

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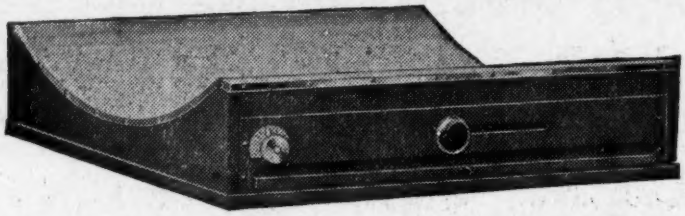

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Fig. 10. Drawing of a section from the region of the tumor, Case 2, showing the atrophic and fibrosed glomeruli and tubules. Infiltrating strands of squamous epithelium with "pearl" formation are also shown.

Fig. 11. Section of the pelvis, Case 2, stained with pyronin methyl green to show the clusters of plasma cells.

SURGERY, GYNECOLOGY AND OBSTETRICS

AN INTERNATIONAL MAGAZINE, PUBLISHED MONTHLY

VOLUME XXXVIII FEBRUARY, 1924 NUMBER 2

SOME RENAL TUMORS[1]

BY SIR WILLIAM I. DE COURCY WHEELER, DUBLIN, IRELAND

President, Royal College of Surgeons in Ireland

IT frequently happens that two or three cases, illustrating rare conditions of the same organs or tissues, are admitted in or about the same time to the wards of a hospital. I recollect that between the years 1910 and 1912 I operated upon three cases of abdominal aneurism, and I never had an opportunity of performing the same operation before or since. In the month of September, Leriche's operation of sympathectomy was performed three times on the femoral and brachial arteries in cases of gangrene, causalgia, and ulcer. No cases suitable for the operation were previously observed.

Within the last year, three consecutive cases of renal tumor came under my care. All three may, I think, in some respects be considered of an uncommon and interesting variety.

It is to these three cases of renal tumors that I venture to call your attention in this communication. I do so with great diffidence, knowing the vast number of renal cases which pass through the great hospitals of America.

CASE 1. Adenomyosarcoma (embryoma.) The patient was a child of 8 months. After birth and for the first few months he appeared normal and healthy, but as time went on the mother noticed that although the appetite seemed good only about half a normal meal could be consumed. There was a gradual loss of weight and co-incidentally the abdomen became enlarged. In consequence of this manifest abdominal distention, advice was sought by the mother in Mercers Hospital, Dublin.

The family and personal history were carefully taken, but were of no significance. There were, in fact, no signs or symptoms of any kind, except for the presence of a large abdominal tumor arising on the left side extending past the mid line toward the right, and becoming lost under the ribs above, and in the iliac fossa below. The child, although slightly emaciated, appeared healthy and well. Urine was passed in normal amounts; there was no history of pain or hæmaturia. (Figs. 1, 2, 3, 4.)

Although the three outstanding signs of renal growth are the presence of a tumor, pain, and hæmaturia, in the tumors of infants the growth is in the kidney and not of the kidney; consequently the tubules, the calyces, and the pelvis become so pressed upon and obliterated at an early stage that no urine can be voided from the affected side. Pain and hæmaturia are, in consequence, absent. As the tumor extends, the renal substance becomes flattened out at the periphery, forming a pseudo capsule comparable to the capsule developed in cases of enlarged prostate, in myomata of the uterus, and in large adenomatous growths of the thyroid gland.

It is possible, in fact likely, that, in the very early stages of development of such a tumor situated in the body of the kidney, hæmaturia may occur, and be the only sign of a central renal growth. At this early period, the calyces, pelvis, and ureter are patent. When the tumor has become palpable and well developed in a child apparently otherwise healthy and strong, there need be no anxiety

[1] Read at the Clinical Congress of the American College of Surgeons, October, 1923.

Fig. 1. Case 1. Adenomyosarcoma (embryoma) in a child of 8 months.

Fig. 2. Photograph of tumor in Case 1, showing the free portion of the kidney.

with regard to the condition of the other kidney.

Cystoscopic and pyelographic examinations, which are always difficult and not without danger in young infants, may as a rule be dispensed with. There is for practical purposes no differential diagnosis in the case of a rapidly growing renal tumor in infants. After careful palpation it will seldom be mistaken for malignant diseases of the retroperitoneal glands, splenic enlargement due to malaria, and leukæmia. Syphilis can be excluded by palpation and by blood examinations, and hydronephrosis in infants is so rare that it need not be considered. About one-third of all cases of malignant growths of the kidney occur in infants in the first 3 years of life, very seldom after 7 years of age. Occasionally a similar tumor affects both sides, and it is difficult to say whether both kidneys are attacked independently, or whether the disease has been primarily in one kidney and that the second has become involved by metastasis. This problem has been investigated in the case of the ovaries. Bilateral malignant disease of the ovaries appears sometimes to arise independently and not by metastasis from one to the other, but it is a difficult matter to prove beyond doubt, and the question is only of academic interest.

When the tumor has grown rapidly to a great size, pressure on the stomach, on the colon, and on the deep veins changes the clinical picture, for now the child suffers from recurrent vomiting, from persistent constipation, from enlargement of the surface veins of the abdomen, and from rapid loss of weight, but nothing abnormal is found in the urine. Up to the point of rapid and great enlargement the silent nature of the tumor formation is impressive.

Mixter (1) said that almost invariably the symptom that brings the child to the doctor is abdominal distention, or the actual discovery of the tumor by the parents; in other words, there may be no early symptoms, but a slight elevation of temperature of from one to three degrees is not uncommon.

The relationship of the tumor appears when the abdomen is opened. The colon comes forward and toward the middle line, and the small intestines are pushed to the opposite side. Removal by the transperitoneal route is not difficult, but a very long incision is essential in a young infant to facilitate the division of adhesions and the rapid dislocation of the tumor from the abdominal cavity. The pedicle as a rule is easily secured and ligatured. In the case under review, the operation was completed within 15 minutes. Operative

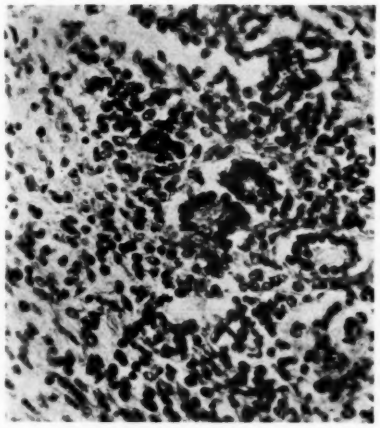

Fig. 3. Photomicrograph, Case 1, showing some of the early attempts at formation of glands.

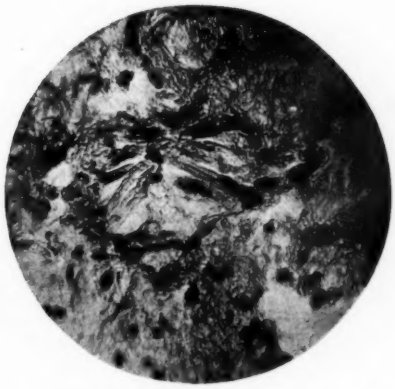

Fig. 4. Photomicrograph, Case 1, showing striped muscle fiber.

rapidity and complete hæmostasis are so important that the lumbar route should never be employed in infants for the removal of large renal tumors.

When the abdomen is opened, access to the tumor is obtained by incising the peritoneum along the outer side of the colon. On the right side care must be taken of the duodenum which lies closely applied in front and may be easily torn. After removal of the tumor, it is a good plan to apply copious layers of cotton wool and complete the dressings by the application of a tight abdominal bandage. The sudden loss of intra-abdominal pressure is compensated for, to some extent, in this way.

The prognosis is extremely poor. Recurrence may be expected in 80 per cent of cases within the first year after operation. If the tumor is found nodular at operation instead of smooth, it may be inferred that sarcomatous changes in the interior have reached the surface, and the outlook may be regarded as bad.

After operation my own case recovered rapidly, but about 6 months later there was a recurrence in the position from which the original tumor was removed, and the child died within a year.

Robins (2) described a case in good health 2 years after operation, but remarks that these tumors are intensely malignant, with a high rate of mortality from operation, and this, with a high rate of recurrence, brings the total mortality rate as high as 93 per cent.

Mixter (3) states that, in nine cases surviving nephrectomy, with one exception all showed rapid recurrence, death occurring in from 4 months to 1½ years. The exception differed in no way, either clinically or pathologically, from other cases of embryoma. The child was alive and well 3½ years after nephrectomy.

Deming (4) discovered a congenital sarcoma in an infant 29 days old. The tumor was successfully removed, and the child was alive and well 12 months afterward.

PATHOLOGY

Naked eye description. The tumor was of an irregular oval shape. It measured 14.5 centimeters in its greatest diameter, and weighed 3¾ pounds. The greater part of its surface was covered by a thin, glistening membrane, serous in character. Where the membrane was absent the tumor appeared to be lobulated, uniformly hard in consistence and of a grey color with areas of a reddish tinge scattered here and there. The remainder (the part covered by the membrane) was smooth, and similar to the exposed portion as regards its consistence and coloration. At one part an elevation was produced by the membrane lying over the kidney which was therefore partly attached to the tumor by this layer. When the membrane was slit up over this area, the kidney was found to be partially embedded in the tumor and its lower pole appeared to thin out gradually and blend with the tumor substance. The amount of kidney tissue which was not embedded was equal to about half the organ. This part was 5 centimeters long and showed the usual lobulations

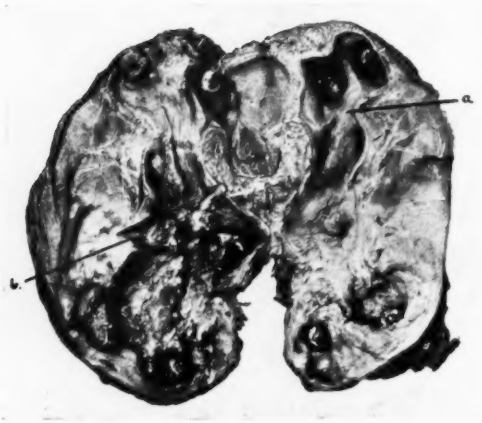

Fig. 5. Case 2. Squamous celled carcinoma with pyonephrosis and stones. *a*, dilated calyces with stones; *b*, showing where the section was made from the pelvis into the tumor mass.

present in young children. When cut in various planes, the tumor was seen to be divided into lobules by fibrous septa. The lobules varied in size, and some areas appeared to be composed entirely of fibrous tissue. As on the surface, red areas were present in places. The consistence and texture of the tissues were similar to that found in uterine fibromyomata. Small areas were noticed which differed in appearance from the surrounding dense tissue; they were of a deeper color and somewhat softer. Compared with the adjacent parts, they formed a contrast similar to that between the mucous membrane and the body tissue of the uterus.

Professor A. C. O'Sullivan kindly examined numerous wax sections made from different parts, and reported as follows:

Microscopic appearance. There was a good deal of variety in the appearances found in different parts of the tumor. In some places there was an arrangement of short spindle cells in a kind of network with clear spaces between. In others, there were long spindle cells with oval or rectangular nuclei arranged in parallel bundles. In others again, there were aggregations of mixed round and spindle cells circular in section, and in the middle of these aggregations a more or less complete attempt at formation of glandular looking structures which, in early stages of their formation, were only distinguished from the surrounding cells by their more regular arrangement. The most remarkable appearance, however, was that of long, narrow fibers running in parallel bundles, containing nuclei of narrow rectangular shape, sometimes quite close together, at other times with a considerable interval between them. These nuclei lay applied to the outside of the fibers. The general appearance of these fibers was not unlike that of a non-medullated nerve fiber, but they stained the characteristic brownish-yellow of muscle fibers with Van Gieson, and in places showed most distinct transverse striation. In the areas mentioned above of networks of spindle cells, one found here and there the same yellowish-brown staining of the bodies of the cells. The vessels were exceedingly thin walled and parts of the tissues showed evidence of œdema—soft and juicy. Between all these various arrangements of cellular material lay sometimes clear spaces, at other places a very fine felted network of fibers, something like neurogliac fibers in their arrangement, but staining bright pink with Van Gieson. The greater part of the tissue consisted of muscle fibers. The appearances are those of the rhabdomyomata described by various writers, and these tumors have now been observed with sufficient frequency to find their way into the textbooks of Ewing and Hausemann.

Fraser (5) found that six different tissues enter into the formation of these tumors: true renal tissue, adenomatous tissue, sarcomatous tissue, non-striped muscular tissue, connective tissue, vascular tissue; and to these may be added a seventh in the present case, striped muscular tissue. He states that an examination of the adenomatous tissue gives convincing evidence that this portion of the tumor has been embryonic in origin.

Case 2. Squamous celled carcinoma. A woman, age 56, was admitted to Mercers Hospital last March. For a number of years she had been treated for bladder trouble with irrigations, etc., and she stated that these troubles commenced with an attack of Malta fever. Twelve years ago she suffered from severe pains off and on in the left lumbar region, but lately, in addition to painful micturition, she noticed that the urine was "muddy" and dark colored, and she came to the hospital for advice.

After routine examination and an analysis of her history, it appeared to us that it was a typical renal calculi case. The urine contained blood cells, pus cells were in abundance, the cystoscope revealed pus shooting through the left ureter, and finally an X-ray photograph demonstrated multiple calculi in the kidney. The photograph showed four stones in the upper pole, ten in the lower pole; the pelvis, ureter, and bladder were free.

The kidney was exposed by the usual lumbar route, and was found so disorganized that nephrectomy was performed.

The pathological report surprised us by stating that, in addition to calculus pyonephrosis, a tumor was present which, on microscopic section, proved to be squamous celled carcinoma. A series of sections demonstrated the transition from pelvic to squamous epithelium with the addition of squamous cells embedded in dense fibrous tissue in the capsular area. Infiltrating strands of squamous epithelium with "pearl" formation were present among the

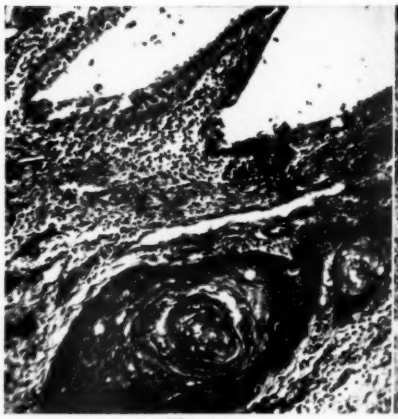

Fig. 6.

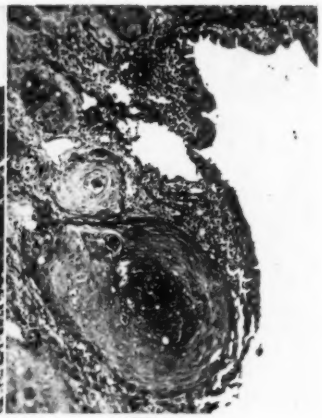

Fig. 7.

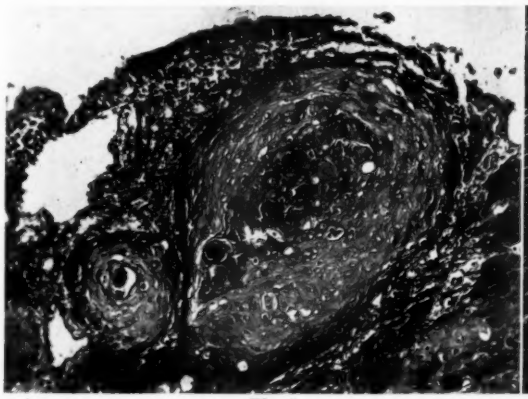

Fig. 8.

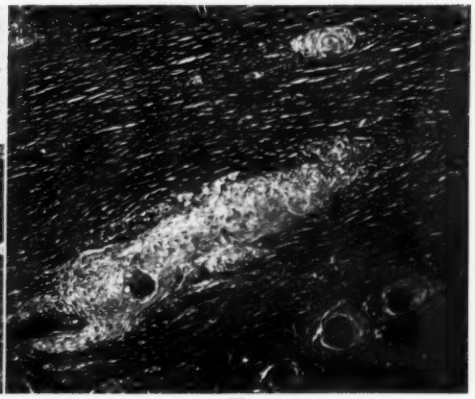

Fig. 9.

Fig. 6. Photomicrograph, Case 2, showing the squamous epithelium lying deep to the epithelium of the pelvis.
Fig. 7 and 8. Case 2. Photomicrographs of the transition from pelvic to squamous epithelium.

Fig. 9. Case 2. The appearance in the capsular area, showing the islands of squamous cells embedded in dense fibrous tissue.

atrophic and fibrosed glomeruli and tubules. A section of the pelvis stained with pyronin methylgreen showed clusters of plasma cells.

From the surgical literature, it appears that squamous celled carcinoma of the kidney in the region of the pelvis is very rare but arises in the great majority of cases in which calculi are already present. It is another example of the development of cancer as a result of prolonged irritation. It may be that a number of cases of squamous celled carcinoma have not been recorded, and that the rarity of the condition is more apparent than real. Neglect in making routine microscopical examinations of specimens removed which, at first sight, appear to be only common examples of calculus nephritis might explain a miscalculation of this kind.

William Mayo (6) states that, of all cases of epithelial cancer of the kidney which came to operation, not less than 50 per cent were demonstrably superimposed on extensive renal calculus formation.

Bugbee also states that the rôle of infection and nephrolithiasis in the etiology of carcinoma of the kidney is suggestive.

Fig. 12. Drawing showing angioma of kidney at *a*.

Graves and Templeton (7) report two cases of combined tumor of the kidney, in which there were neither calculi nor evidence of chronic pyelitis.

Wells (8) referring to squamous celled carcinoma of the kidney pelvis, states that the formation of keratinising squamous celled carcinoma in the renal pelvis is a rare occurrence; and in a case reported by him the metaplasia of the transitional epithelium to the squamous form was apparently the result of chronic irritation from renal concretions.

Newman (9) records that squamous celled carcinoma is very rare, and refers to a case published by Kundral and Halle, who suggested the idea that these growths are plaques of leucoplakia which have, as a consequence of local irritation, taken on malignant action. The irritation may be the presence of a stone in the pelvis or bacterial infection. The tendency of these growths is to bleed; in fact, it is the rule, so that hæmaturia is the only early indication of the disease.

According to this Newman authority, when the ureter becomes excluded by clot, or if its walls become involved in the disease, other symptoms develop, such as renal pain and swelling. He had not seen any cases of squamous celled cancer of the renal pelvis, and after a careful search of the literature he was able to discover only nine properly recorded cases.

In most, if not all, of the few cases of squamous celled carcinoma reported in the literature, the transition from one epithelium to the other appears to have been directly brought about by the presence of infection and of stones.

Cumming (10) discusses leucoplakia of the renal pelvis and the metaplasia resulting in the formation of stratified epithelium. Dr. McCarty of the Mayo Clinic tells me that in 1,648 kidneys which came to operation in 10 years 7 epitheliomata were found.

CASE 3. Angioma. (Fig. 12.) Last March a clergyman, age 57, without previous warning or any signs of ill health, developed a severe urinary hæmorrhage.

There was no pain, tenderness, or other indication of the site of the bleeding. The urine passed had the appearance of pure blood. Attempts at cystoscopy failed; it was impossible to free the bladder of blood, even for a moment, and the examination ended without having ascertained whether the bleeding was from the bladder or of renal origin. X-ray photographs were taken with negative results. There was no pus and no micro-organisms in the urine, and renal growth was suspected.

An effort was made to check the hæmorrhage by the administration of horse serum, morphia, calcium, and by blood transfusion, with only partial success. On the sixth day the urine was much clearer, but after palpation of the left renal region the patient stated that he felt that the hæmorrhage was commencing again, and red blood was almost immediately passed from the bladder. It was now evident that palpation had excited the hæmorrhage.

The patient was by this time suffering severely from loss of blood and, notwithstanding blood transfusion, there was profound anæmia, and an operation was undertaken with some anxiety. The kidney was found enlarged and cyanosed; the pelvis was distended with blood clot, but the source of the hæmorrhage in the kidney was impossible to ascertain. The pedicle was clamped, ligatured, and the kidney rapidly removed. Recovery was uninterrupted, and the patient is now well.

On examination of the specimen, small scarlet or purplish clusters of vessels, as described by Virchow, were found in the renal tissue immediately under the

capsule. Under the mucous membrane of the pelvis an angiomatous tumor resembling renal varix was discovered but was due to a proliferation of small and medium sized blood vessels forming a rich arterial plexus.

In this case, it was possible to see the blood, on cystoscopic examination, flowing in considerable quantity from the left ureter On section an angioma was found occupying the third uppermost calyx, but it could not be seen when the organ was *in situ*, although it was bleeding freely.

Newman records a very similar case of angioma of the renal pelvis with hæmaturia, no X-ray shadow, no enlargement of the kidney, bacteriological examination negative, and nephrectomy with good result.

Swan (11) describes a case of sudden profuse hæmaturia, without any preceding pain. The kidney was removed and divided in length, when a spongy mass was seen in the central part oozing blood freely from the surface. On microscopical section, the renal tissue was found infiltrated with blood around an area showing the structure of an angioma.

Sir Henry Morris's (12) book records the postmortem finding of multiple angiomata of the left kidney, one of which had ulcerated into the upper calyces. To the naked eye, the cut surfaces showed the open mouths of two or three large vessels with a wide zone of cavernous tissue surrounding them. Microscopically, these areas were composed of a collection of vascular spaces, as in an angioma.

Sennels (13) reports two cases of renal angioma. In the second case a malignant papilloma of the bladder was subsequently found and the nature of the original tumor of the kidney is, therefore, open to some doubt. The first case resembled very closely the case now reported. The absence of pus and bacteria and the free bleeding led to the diagnosis of tumor.

PATHOLOGICAL REPORT

CASE 3. Angioma of the kidney. The hæmorrhagic areas on the surface were seen to be in the tissue of the kidney and not between it and the capsule. Toward the upper pole was a yellow nodule the size of a small pea, which was found to be composed of tissue similar to that of the suprarenal cortex and was apparently a small adrenal "rest." This finding was a coincidence and had no significance. The pelvis was found full of blood clot and the lining was blood-stained. At the upper end of the pelvis a plexiform mass of thick walled vessels was observed occupying an area of about 1.5 centimeters in diameter. In the hæmorrhagic regions the renal epithelium had undergone very extensive degeneration. Some hyaline glomeruli were present. Many of the tubules were packed with blood cells and extravasations into the surrounding tissue were numerous. The plexus was composed of thick walled arteries.

A difficulty arises in cases of renal angiomata. The condition is only discoverable after nephrectomy. The bleeding might equally well arise without pathological change in the renal tissues, and be the so called "essential hæmaturia" which can be treated successfully by decapsulation and other conservative methods. It is noteworthy that, in the recorded cases, the left kidney was affected, and that the angiomata in each instance were placed in much the same situation.

In the Mayo Clinic to date only one case of angioma of the kidney has been observed. This was verbally reported by Dr. McCarty.

I am indebted to Dr. E. C. Smith, Pathological Laboratory of Trinity College, for the pathological reports and for the illustrations.

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PRIMARY SARCOMA OF THE PENIS

Report of a Case with a Review of the Literature

By JAMES J. JOELSON, M.D., Boston
From the Urological Clinic of the Peter Bent Brigham Hospital

ON September 5, 1922, a Swedish laborer, 57 years of age, was admitted to the clinic giving the following history:

Two years ago he first noticed a small wart-like growth on the left side of his glans penis, near the corona. This he cut off himself with a pair of scissors. He did not notice anything further until 8 months before admission, when a small reddish nodule appeared at the site of the original wart. This grew quite rapidly, and in 3 months had reached a size of about 3 centimeters in diameter. He went to a local physician who cut the growth off under local anæsthesia, without any further treatment. The tumor soon began to grow again and in 3 or 4 months had reached its previous size. He now went to another physician who excised a small piece of tissue for microscopic examination and sent it to a laboratory where a diagnosis of sarcoma was made. Following this biopsy, which was done 6 weeks before admission to the hospital, the tumor grew very rapidly, gradually involving practically the entire glans penis and pushing the external urinary meatus far over to the right. At no time had he had any difficulty in urinating. He had lost no weight or strength and had been working every day until admission.

His family and past histories were irrelevant. There had been no previous history of injury or phimosis. He denied venereal disease.

Examination of the penis revealed a large, red, fungating, ulcerated tumor which had involved the entire glans and pushed the prepuce back. The tumor was 6 to 7 centimeters in diameter. A yellowish, foul discharge was present. The urinary meatus had been displaced entirely over to the right side so that the urinary stream, which was moderately decreased in caliber but forceful, came from the region of the corona. The shaft of the penis appeared normal and showed no evidence of extension of the growth. The inguinal nodes on both sides were definitely enlarged. (Fig. 1.)

General physical examination was negative and together with the X-ray examinations revealed no evidence of metastasis to the lungs, ribs, pelvic bones, or long bones.

The blood Wassermann was negative; the hæmoglobin 85 per cent, the red count 4,500,000.

On September 9, 1922, under nitrous-oxide and oxygen anæsthesia, a dissection of the inguinal nodes and an extirpation of the penis with transplantation of the urethra was done after the method described by Cunningham.

The patient had a very smooth postoperative course. A slight amount of wound infection occurred, but this was readily controlled. He was discharged in good condition about 3 weeks after operation.

Since his discharge the patient has been followed very carefully. He is doing very well, has no complaints, and has gained some weight. The perineal urethral orifice is not strictured, and urination is unimpaired. When last seen (May 12, 1923), 8 months after operation, there was no evidence of recurrence or metastasis to be found clinically or by X-rays of the lungs, skull, entire spinal column, ribs, pelvic bones, and all the bones of the extremities.

Pathological report. The following is Dr. S. B. Wolbach's description of the specimen. Gross description: Specimen consists of several masses of fat tissue, removed from the groin, and a penis. Palpation of the fatty tissue reveals several discrete, firm masses measuring up to 1 centimeter in diameter. These are apparently lymph nodes. On section the nodes are somewhat firmer than normal and are greyish in color with here and there a white, firm area, probably metastases. Fixed in Zenker.

The penis with contained tumor weighs 138 grams and measures 12 centimeters in length. Surrounding the glans penis so that it is not discernible is a large, fungating tumor mass 7 centimeters in width and extending back over the penis 3.5 centimeters. The tumor mass is made up of irregular, nodular areas varying from 0.2 to 1.5 centimeters in diameter. The color varies from a reddish grey to a sort of greenish grey in the center of the tumor mass. It is very soft, friable, and in areas is covered by a greyish fluid. It has an exceedingly foul odor. The skin immediately adjacent to the tumor is markedly thickened. The remainder of the penis appears normal. Several small portions of the tumor are taken for microscopic diagnosis; the remainder is preserved in Kaiserling solution as a gross specimen. (Figs. 2 and 3.) Microscopic examination: Six sections of lymph nodes show normal germinal centers and lymph sinuses. There is an increase of fibrous connective tissue in one section. There is no evidence of tumor metastasis.

There are sections from various portions of the tissue and penis stained with eosin methylene blue, phosphotungstic acid hæmatoxylin, aniline blue connective tissue stain, and Van Gieson's stain. The tumor as a whole is fairly uniform in structure. The cells are for the most part elongated, fusiform, or spindle shaped. The special stain shows in most places traces of collagen between the cells, and in a few places the collagen is fairly abundant. It is

Fig. 1. Appearance of the lesion on application of the patient to the clinic. The tumor was a large, red, fungating mass, pushing back the prepuce.

Fig. 2. Side view of the gross specimen, actual size, showing the sarcoma involving practically the entire glans and pushing back the prepuce.

evident that the tumor is growing rapidly, as there are many mitotic figures. There are also numerous multipolar mitoses. Just beneath the ulcerated surface, the tumor cells take a number of shapes, round and ovoid on cross section, with rather large nuclei. The tumor cells here appear somewhat necrotic and are invaded by some polymorphonuclear leucocytes. At the base the tumor is fairly sharply outlined, but close inspection shows that its peripheral portion consists of elastic tissue derived from the tunica, and careful examination shows it definitely invading. In the mass of tumor itself there is no recognizable normal tissue except blood vessels that are completely surrounded by the tumor and which show infiltration of their walls by tumor. There are several cross sections of the corpora cavernosa posterior to the tumor, including the dorsal vessels and lymphatics. There is no evidence of tumor in these sections. (Figs. 4, 5, 6.) Diagnosis: Fibrosarcoma of glans penis.

REVIEW OF REPORTED CASES

A careful search of the literature has revealed only thirty-five cases of sarcoma and endothelioma of the penis. Of these, one is reported as a "fibro-cellular tumor," two as fibrosarcomata, two as spindle cell sarcomata, four as mixed cell sarcomata, seven as round cell sarcomata, eight as melanosarcomata, and nine as endotheliomata. Two are not classified. The first of these is the case of Mr. Seymour Sharkey who reported having seen a case of sarcoma of the penis but remembered none of the details other than that it had been clinically diagnosed as an epithelioma and was a spindle cell sarcoma. The other is Podrazki's case which is mentioned briefly by Jacobson. This patient lived only a year after the onset of the tumor and had inguinal gland metastasis. Unfortunately, Jacobson does not give the reference to the original article and I could find no mention of it anywhere else.

Weir reports a case of "rare form of cancer of the penis" which clinically appears to be a sarcoma, but histological diagnosis is wanting. Si-Mohamed's article is entitled "Sarcoma de la verge" but his histological report is merely "lobulated epithelioma" without any further description. Vopel reports three

Fig. 3. Sagittal view, actual size, of distal portion of the penis. The sarcomatous involvement is seen to be mainly limited to the glans although it slightly invades the distal ends of the corpora cavernosa.

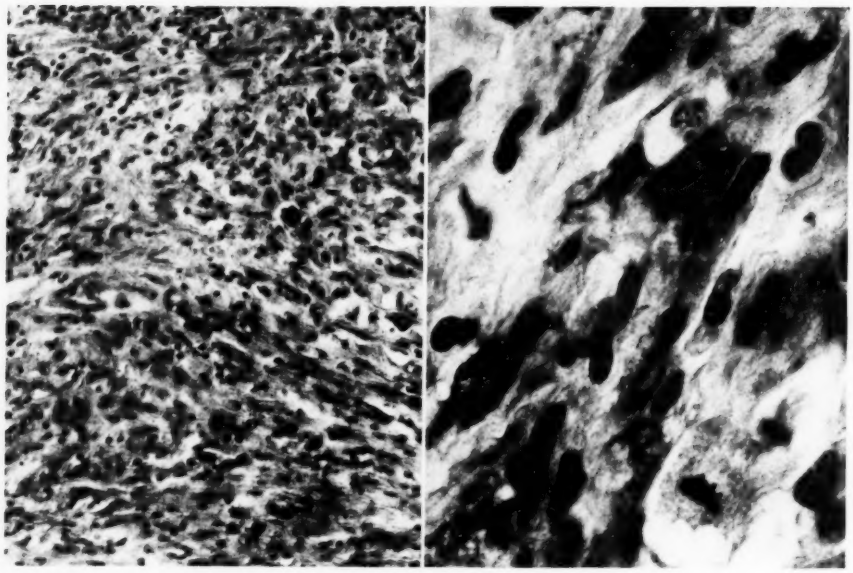

Fig. 4 (at left). Showing general structure of tumor, which as a whole was quite uniform. × 200.
Fig. 5. Showing the elongated spindle shaped type of cell. A mitotic figure is seen. These were numerous and frequently multipolar. × 1038.

cases of sarcoma of the penis, but in only one of these (Case 3) is there sufficient evidence to tabulate it among the reported cases. In the other two cases the prostate was so extensively involved in the tumor mass that the author leaves the question open as to whether or not the tumor was primary in the penis. In addition there are no satisfactory histological reports in these two cases.

The great majority of these cases occurred in men of the "cancer age," but four occurred in younger adults, one in a boy of eight, and two in infants. The most common subjective symptoms were pain in penis or perineum, dysuria, and occasionally acute retention of urine. The majority of the cases of endothelioma had "priapism" with an enlarged penis as if in semi-erection.

As a general group, sarcoma of the penis is a very malignant tumor, as is indicated by the following figures. Unfortunately the "follow-up" period had been very short at the time when most of these cases were reported and therefore the number of 3 or 5 year cures cannot be determined.

	Cases	Total
a. Duration in patients who were living at the time case was reported—		
Living 11 to 15 years after onset	1	
Living 6 to 10 years after onset	1	
Living 1 to 5 years after onset (One case has recurrences)	5	
Living 6 months to 1 year after onset (One case has recurrences)	2	
Living less than 6 months after onset	0	9
b. Duration in patients who were dead at the time the case was reported—		
Lived 11 to 15 years after onset	0	
Lived 6 to 10 years after onset	2	
Lived 1 to 5 years after onset	4	
Lived 6 months to 1 year after onset	8	
Lived less than 6 months after onset	4	18
c. Cases in which total duration was not stated		9
Total		36

Tables I, II, III, and IV summarize the reported cases obtained from the literature.

The cases in Table I are less malignant than any of the other groups. These tumors do not metastasize freely but do tend toward

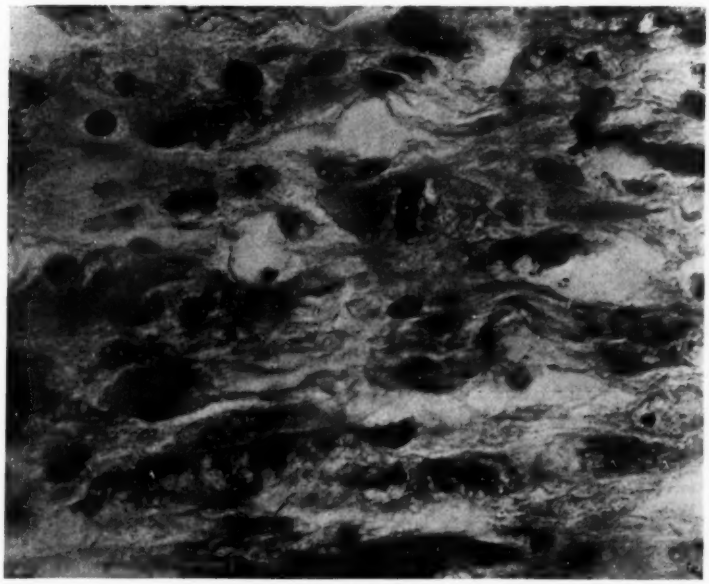

Fig. 6. Van Gieson stain. The waxy fibrillary intracellular substance is revealed. A mitotic figure appears. × 1250.

local recurrences. The majority of them take origin from the corpora cavernosa, less often from the sheath of the corpora than from the erectile tissue. However, two take origin from the glans penis. Eight of these cases had a simple excision of the tumor done first, but in all of these, except one, a more radical procedure such as amputation or extirpation was later necessary because of local recurrences.

The round cell sarcomata (Table II) are more malignant than the previous group and metastasize earlier and more freely. The great majority of these also started in the corpora cavernosa.

The melanosarcomata (Table III) also metastasize freely, but, as is indicated by the longer duration before operation, do not grow as rapidly as the round cell sarcomata. In all of these cases the tumor takes its origin at the distal end of the penis; either the glans or the prepuce.

The endotheliomata (Table IV) form the largest single group. They appear to be very malignant and rapidly growing tumors.

SUMMARY

A case of fibrosarcoma of the glans penis of 2 years' duration is presented. Two local excisions and a biopsy had been performed. No evidences of metastases were apparent. Eight months after complete extirpation of the penis with dissection of both groins there was no evidence of recurrence or metastases.

Thirty-five cases of sarcoma of the penis are collected from the literature and are tabulated. Of the entire series 9 cases fall into the group generally known as endotheliomata, the most malignant type. Seven fall into the group of round cell sarcomata, also a very malignant type. Eight fall into the general group of melanosarcomata, a definitely malignant but more slowly growing tumor. Nine of the collected cases appear to be spindle or mixed cell sarcomata and tend to have a relatively low degree of malignancy. Our case belongs to this latter group.

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TABLE I.—CASES REPORTED AS FIBROSARCOMATA, SPINDLE CELL AND MIXED CELL SARCOMATA

Reported by	Pathological diagnosis	Age	Point of origin	Metastasis	Duration before operation	Type of operation	Result	Total duration	Symptoms and remarks
Hutchinson 1854	"Fibro-cellular tumor"	8	Corpus cavernosum	None	10 wks.	Local excision	Living without recurrence at 5 months after operation	7½ mos.	Rapidly growing tumor of penis in the region of the base of the glans. Had caused several attacks of acute retention. Local excision; base of tumor cut through without any attempt at enucleation. Histological report merely states that it was "an ordinary example of a fibrocellular tumor."
Beck 1872	Fibrosarcoma	56	Corpus cavernosum	Local recurrence	13 yrs.	1. Circumcision 2. Amputation 2 years later	Living Recurrence 2 years after first operation	15 yrs.	No symptoms. Wart-like growth beneath prepuce close to base of glans which was removed by a circumcision. Recurrence in the scar requiring an amputation 2 years later.
Péraire 1919, 1920	Fibrosarcoma (spindle cell)	43	Corpus cavernosum	Local recurrence	1½ yrs.	1. Local excision 2. Extirpation of penis 14 mos. later	Living Recurrence 14 mos. after first operation	2½ yrs.	Marked pain for 3 mos. Tumor easily enucleated at first operation, but extensive recurrences in both corpora cavernosa required a complete extirpation of the penis 14 months later.
Porter 1880	Large spindle cell	44	Corpus cavernosum (probably from sheath)	None	6 mos.	Excision	Died one week after operation	6 mos.	Very rapidly growing tumor in the region of the root of the penis. Excised tumor weighed 1½ pounds. Died of postoperative complications.
Galt 1911	Spindle cell	60	Corpus cavernosum	Inguinal lymph nodes. Clinical evidence of abdominal metastasis	8 mos.	Extirpation of penis and dissection of groins.	Died 7½ weeks after operation	10 mos.	Started as a small papule on the dorsum of the penis and grew rapidly.
Joelson 1923	Fibrosarcoma (spindle cell)	57	Glans penis	Local recurrence	2 yrs.	1. Local excision 2. Local excision 3. Extirpation and dissection of groins	Living at 8 mos. after operation without recurrence or metastasis	2⅔ yrs.	No symptoms other than tumor. Excision 2 years before admission; recurrence 16 months later. Second excision 5 months before admission. Rapid recurrence. Extirpation finally done 2 years after first appearance. No clinical or X-ray evidence of metastasis 8 months after last operation.
Fenwick 1889	Round and spindle cell	36	Sheath of left crus	Local recurrence	3½ yrs.	1. Local excision 2. Excision of left crus	Living 2 years after first operation. No recurrences 6 months after second	5½ yrs.	4 yrs. intermittent pain in perineum and a perineal tumor for 3½ yrs. Extensive recurrences when seen 20 months after local excision. No recurrences at 6 months after second operation.
Battle 1892, 1899	Spindle and round cell	50	Left crus	Clinical evidence of metastasis to lungs and left iliac fossa	2 yrs.	1. Excision of tumor 2. Excision of recurrences 4 years later	Died 5 years after first operation from metastases	7 yrs.	Difficulty in micturition and appearance of tumor 2 years before operation. Recurrences occurred in scar 6 months after a wide excision requiring a second operation which was done 4 years later.
Hildebrand 1898	Round and spindle cell								The author briefly mentions this case at the end of his article on endothelioma but gives practically no details.
Key 1903	Round and spindle cell (pigmented)	74	Glans penis	Local recurrence	4 yrs.	1. Excision 2. Amputation	Living without recurrences 3 years after amputation	7 yrs.	Small ulcerated tumor excised 4 years before seen by Key. Recurrence in 6 months and amputation done 3½ years later. There is no recurrence or metastasis at 3 years after amputation. Author considers it a sarcoma with hæmosiderin and hæmatoidin pigment in it and not a melanosarcoma.

TABLE II.—CASES REPORTED AS ROUND CELL SARCOMATA

Reported by	Pathological diagnosis	Age	Point of origin	Metastasis	Duration before operation	Type of operation	Result	Total duration	Symptoms and remarks
Koehler 1880	Round cell	21	Corpora cavernosa	Lungs, liver, prostate, inguinal nodes, ribs, skull, and between dura periosteum of spinal canal	4 mos.	Local excision	Died 4 mos. after operation of extensive metastases	8 mos.	Difficulty of urination and growing tumor in posterior portion of scrotum of 4 months' duration. Enlargement of inguinal nodes noticed 2 months after onset. Local excision of a "tumor of the size of an infant's head." Immediately after operation multiple metastases appeared all over body.
Battle 1885	Round cell	60	Glans penis	None	10 mos.	Amputation	Died 43 days after operation	11¾ mos.	Patient noticed a gradual painless enlargement of the penis with slight bleeding. Died 6 weeks after operation, the last symptoms being thirst, jaundice, hiccough, and signs of exhaustion. No clinical evidence of metastases.
MacCormac 1895	Round cell (small)	64	Corpus cavernosum	None	6 mos.	Complete extirpation of penis and castration	Died on sixth day after operation	6 mos.	Hard tumor, about the size of a fist, situated in the upper part of the right scrotum and extending backward to region of perineum.
Vopel (3rd case) 1898	Round cell	41	Corpus spongiosum (connective tissue of urethral mucosa?)	Inguinal lymph nodes	6 mos.	Amputation and dissection of both groins	Recovered from operation but no further facts given		Swelling appeared in penis several days after injury. Soon began to have difficulty in micturition requiring catheterization. Colmers considers this to be an endothelioma.
Pupovac 1900	Round cell	47	Corpora cavernosa	Inguinal lymph nodes	6 mos.	Amputation. Dissection of both groins and laparotomy for retroperitoneal glands	Died fourth day after operation	6 mos.	Hard, painless swelling in pendulous portion of urethra of 6 to 7 months' duration. Patient died fourth day after operation of acute peritonitis.
Webber 1905	Round cell (large)	51	Corpora cavernosa	Inguinal lymph nodes. Recurrence in remaining portion of penis and growth projecting from suprapubic wound	6 mos.	Suprapubic cystotomy. Amputation and castration	Living at 5 months after operation, but in very poor condition	11 mos.	Pain and difficulty on urination for 1 year and tumor for 6 months. Tumor obstructed catheter. Amputation done but at 5 months patient is very weak, anæmic, and bedridden, with recurrences.
Heigel (2nd case) 1913	Round cell (large)	32	Corpus cavernosum	Inguinal lymph nodes	1½ yrs.	Complete extirpation of penis and dissection of both groins	Not stated		Started as a pea-sized tumor on dorsum of penis which gradually increased in size and the inguinal lymph nodes later became enlarged.

TABLE III.—CASES REPORTED AS MELANOTIC TUMORS

Reported by	Pathological diagnosis	Age	Point of origin	Metastasis	Duration before operation	Type of operation	Result	Total duration	Symptoms and remarks
Holmes 1871	Melanosarcoma (spindle cell)	52	Glans penis (urethral mucosa?)	Inguinal lymph nodes (?)	8 yrs.	Amputation	Recovered from operation but no further details given		Slight dysuria. Began as a small, hard, black spot on the glans. Induration extended along course of urethra. Slightly enlarged inguinal nodes.
Gould 1880	"Melanotic epithelioma"	75	Glans penis	None	5 yrs.	Amputation			Dysuria. Slowly growing black tumor on glans around which several small tumors had formed.
Murchison cit. by Gross	Melanosarcoma	54	Prepuce	"Liver, pleura, lymphatic glands, and other structures"		None	Died 2 yrs. after onset with metastases	2 yrs.	Began as a small excrescence on the prepuce. from where it gradually extended to the glans. Was almost black in color, bled readily when touched and was the seat of great pain.
Golding Bird 1885	Pigmented sarcoma (spindle cell)					Amputation			This case is briefly mentioned by the author at a meeting of the Pathological Society. No further details are given other than that the patient recovered from the operation.
Fisher 1887	Melanosarcoma (round and spindle cell)	53	Glans penis (urethral mucosa?)	Inguinal lymph nodes and dorsal lymphatics of penis. Recurrences in pubic scar and in groin	9 mos.	Amputation and dissection of both groins	Living 7 mos. after operation but has rather extensive local recurrences	16 mos.	Difficulty in urination, weak stream. Tumor began as bluish spot on glans. At operation metastases seen in groin outside of lymph nodes and adherent to and growing into veins.
Miner 1896	Melanosarcoma (spindle cell)	55	Glans penis (erectile tissue)	Liver	8 yrs.	Amputation	Died 1 week after operation	8 yrs.	Began as a bluish discoloration on glans and gradually grew and ulcerated. No symptoms. Pigmented ulcerated tumor on glans and enlarged nodular liver. Autopsy showed metastasis only to liver. A large amount of brown purulent fluid was present in peritoneal cavity.
Payr 1899	Melanosarcoma	63	Glans penis	Inguinal lymph nodes, liver, lungs and abdominal wall	2 yrs.	1. Local excision. 2. Amputation and dissection of both groins	Died 4 mos. after operation with multiple metastases	2⅓ yrs.	Started as small wart on glans, which was excised. 6 months later a pigmented tumor appeared at the same site and grew rapidly. Had difficulty in urinating for several weeks and acute retention at time of admission.
Peters 1922	Melanosarcoma	72	Glans penis	Inguinal lymph nodes. Pelvic metastasis. Local recurrence at amputated stump	16 mos.	1. Amputation 2. Dissection of both groins	Died 5 mos. after operation with metastases	21 mos.	Patient refused to allow groin dissection at first operation. Three months later had atresia of urethral orifice due to recurrences and orifice was reopened and groins dissected.

TABLE IV.—CASES REPORTED AS ENDOTHELIOMATA

TABLE IV.—CASES REPORTED AS ENDOTHELIOMATA

Reported by	Pathological diagnosis	Age	Point of origin	Metastasis	Duration before operation	Type of operation	Result	Total duration	Symptoms and remarks
Maurer 1883	Endothelioma	50	Corpora cavernosa	Inguinal lymph nodes. Many distant metastases to skin and subcutaneous tissues		None	Died 4 mos. after onset	4 mos.	Very rapidly growing tumor. No symptoms. Penis enlarged as if in semi-erection. No change in shape.
Tripke 1897	Endothelioma	16							Author gives a pathological report but practically no clinical facts. Borst also mentions this case.
Alexander and Dunham 1897	Endothelioma	50	Corpora cavernosa	None mentioned	6 mos.	Excision	Died 2½ yrs. later; cause not known	2 yrs. and 10 mos.	Painless lump on dorsum of penis extending equally into both corpora cavernosa causing no symptoms. Colmers considers this an endothelioma of the lymph spaces rather than of the cavernous sinuses.
Hildebrand 1898	Endothelioma (?)	68	Corpus spongiosum	None	1 yr. +	Excision	Living at ¾ yr. after operation without recurrence	1½ yrs. +	Lump on under surface of pendulous portion of penis. Only symptom was terminal dribbling. Hildebrand brings up the differential diagnosis with carcinoma of the penis but favors endothelioma. Colmers and Borrmann consider carcinoma the more likely diagnosis.
Colmers 1903	Endothelioma	56	Corpus cavernosum or spongiosum	Heart, pericardium, lungs, and inguinal lymph nodes	2 mos.	Amputation	Died 1 week after operation	2 mos. +	Painful priapism. Dorsal vein of penis found to be invaded by tumor.
Creite 1905	Endothelioma (?)	2 yrs. and 2 mos.	Corpus cavernosum or spongiosum	None	8 days +	Amputation	Died 1½ mos. after operation	2 mos. +	Penis three times larger than normal; erect; dilated veins on dorsum. Creite brings up the differential diagnosis between endothelioma and carcinoma and favors the latter. Schultze considers it to be an endothelioma.
Migliorini 1905	Perivascular sarcoma	50		None clinically	1½ yrs.				Author gives very few clinical details, as he merely made a pathological examination of a piece of tissue sent to him by a colleague.
Pelagatti 1910	Endothelioma	65	Corpora cavernosa	None clinically		None	Died 7 mos. after onset	7 mos.	Marked dysuria and difficulty of urination. Severe pain in penis radiating to perineum. Patient was admitted with acute retention and uræmia. Catheterized without difficulty. Died 4 days after admission.
Gobbi 1922	Endothelioma	10 mos.	Corpora cavernosa	Inguinal lymph nodes		None	Died 90 days after onset	3 mos.	Marked enlargement of penis, which was firm as if in erection.

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PATHOGENESIS AND TREATMENT OF SO-CALLED CONGENITAL CEREBRAL HERNIÆ[1]

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SO-CALLED congenital cerebral herniæ are generally classified as encephalocele and exencephaly, the difference between these two conditions being rather a quantitative one.

In exencephaly a larger or smaller portion of the brain substance becomes dislocated through an opening in the cranium. It may be looked upon as an ectopy of the brain and is but of a teratologic interest, as it is incompatible with life and the surgeon is seldom called upon to deal with this morbid condition.

Encephalocele might be defined as a tumor-like formation originating in the brain and its meninges. Both are rare diseases, the statistics showing but two or three cases in 10,000 newborn children.

The most frequent location of the cerebral herniæ is in the mesial line of the cranial vault, in the frontal or occipital region. Herniæ of the base are quite rare, while lateral herniæ are said not to exist at all. These are rather pseudo-traumatic formations occurring during labor, for instance.

REPORT OF CASES

Case 1. A girl, 11 months old, of good mentality, was brought to a military hospital with a tumor in the occipital region of the skull. The tumor was round and occupied the external occipital tuberosity and extended down the spine. It had a pedicle 2 centimeters long. The skin covering the newgrowth was thin, could be folded, and was covered with thin hair which was quite abundant over the rest of the head. The tumor was transparent to light, quite painful on pressure, but could not be reduced in size by compression, nor could it be pushed back. The child could not lie on its back on account of pain. Puncture yielded a transparent, pale yellow fluid which was not studied in detail. An operation performed 9 days after the examination revealed a bone defect in the occiput below the external tuberosity, 1 to 1.5 centimer in diameter. In the center of the gap there was an opening of 5 millimeters. This was covered with a solid shiny band extending from the skull cavity to the tumor. The latter was removed and the band replaced into the cranium. The postoperative course was marked by an elevated temperature and abundant discharge of the cerebrospinal fluid. On the seventh day the temperature became normal and on the eleventh day after the operation the child was discharged in a good condition. When seen 6 months later it was perfectly well. The pathological examination of the removed tumor showed it to be thin walled containing fluid and some coagula. The cavity of the pedicle had a polyp-like thickening which consisted of brain and connective tissue (blood vessels, dura, and pia). Neuroglia was found around the blood vessels which formed plexuses and somewhat resembled ependymal cells. The brain tissue was dotted with numerous nuclei of various sizes and form. In some places the neuroglia invaded by connective tissue fibers contained blood vessels, capillaries and perivascular spaces.

The superficial layers of the pedicle showed inflammatory phenomena (leucocytes, fibrin). The inner surface of the cyst was represented by the meninges, especially the dura, which was richly vascularized. The epithelium of the skin was thinned, the papillæ obliterated, the stratum corneum very much atrophied and in some places entirely absent. Sudoriferous and sebaceous glands were in some instances much altered and atrophied. The coagula consisted of fibrin enclosing some leucocytes.

The foregoing polypous thickening is to be looked upon as a glioma (ependymal), most likely as a result of maldevelopment, while the presence of ependymal cells indicated that they became disclocated in the embryonic life of the patient. In addition, one should take into consideration the following factors: (1) disturbances of the lymph and blood circulation, as the result of the disorder of the physiological equilibrium of the tissues; (2) defects in the meninges which control the cerebral circulation; and (3) the increased intracranial pressure. This last might be the result of increased secretion of the spinal fluid by the choroid plexus because of its disturbed blood circulation. It may result in a serous meningitis, accumulation of lymph in the subarachnoid space, and ultimate formation of a meningeal cyst. Being constantly filled with fluid, it increases in size, the walls becoming tense, thinned, and atrophied. These factors often cause a rupture of the tumor.

Mesodermal changes consisted in proliferation of connective tissue and blood vessels, both replacing the neuroglia tissue.

In short, the etiological factors in this case were (1) dystopy of the brain elements; (2) incomplete closure of the skull because of the mechanical obstacles brought on by the dystopy; (3) protru-

[1] Presented before the Society of Russian Surgeons in Moscow, February 12, 1923.

sion of the meninges with formation of a meningeal cyst, partly because of disturbances of lymph and blood circulation and the consequent increased intracranial pressure.

The diagnosis in this case is hydrocephalo-meningocele occipitalis inferior.

CASE 2. Boy, 2½ years old, a full term child, entered the hospital with a large tumor over the base of the nose. At the time of birth it was the size of a button, and located on the side of the nose. It was gradually getting larger.

Examination showed a well developed boy, unable to speak. On the face there was a somewhat tuberous tumor attached to the nasal bones. It was soft, at some points transparent, at others pulsating. The base was broad and fixed. Compression did not reduce the tumor nor did it influence or affect the radial pulse. It was, however, very painful. The skeletal bones as well as the skull showed no abnormalities. An operation performed 8 days after the admission showed that the tumor located at the internal region of the orbit was separated from the bones. The nasal bones at their base were broken through and turned upward. The tumor which extended into the skull cavity could not be removed and was resected at the base. Child died 3 days after the operation.

Microscopic examination. The surface of the tumor was represented by a normal skin containing glands and papillæ. The corium exhibited large quantities of unripe striated muscles and lymphatics. Foci of neuroglia tissue were present around the sudoriferous glands. The further from the epidermis the more were the muscle and connective tissue replaced by nerve tissue which was lobular in structure. The interlobular spaces were invaded by cellular connective tissue, muscle fibers, and blood vessels. The latter were compressing the neuroglia which showed as small foci scattered in the mass of mesodermal tissues. The lobules themselves were invaded by connective tissue which in its turn formed lobules. A specimen from the center of the tumor mainly showed neuroglia tissue invaded and in many instances replaced by connective tissue, though not so intensely as in the previous case. Muscles were absent, but vessels and even capillaries were quite numerous in the interlobular areas of connective tissue. In another specimen, again, neuroglia predominated and was richly vascularized containing numerous newly formed capillaries.

The foregoing growth should be looked upon as a dystopy, that is, as the result of incomplete separation of the corneal layer from the medullary tube. The consequence of the dystopy of the brain tissue was a defect in the cranial bones, a deficient growth of glial tissue which ultimately became replaced by connective tissue.

The study of cases described by others shows that the older the tumor (cephaloma) the more connective and less brain tissue there is to be found. In fact old cephalomata consist principally, if not exclusively, of connective tissue, while young cephalomata, in a fetus, for instance, consist of brain tissue with a tendency of the latter to be replaced by connective tissue, as shown also in the second case.

With Lissenkoff and Petroff, I admit that meningocele as such does not exist, that is to say, a mere protrusion of cerebral meninges with a cyst formation filled with cerebrospinal fluid is but a secondary manifestation, the primary phenomenon being dystopy of the brain. Those cases of meningocele in which no traces of brain elements could be found at all should be explained as encephalomeningocele where the brain tissue disappeared because of secondary invasion of connective tissue.

The following points should be emphasized: (a) the principal location at the root of the nose and on the occiput; (b) the cause of the basal cranial hernia which is incompatible with the theory of dystopy due to incomplete and irregular occlusion of the cerebral tube; (c) the cause of dystopy of the medullary tube.

a. Occlusion of the medullary tube begins in the mesencephalon (middle cerebral vesicle) extending caudad and cephalad; the frontal and occipital portions, however, close much later. Therefore, there are greater chances for abnormalities to take place, such as delayed or incomplete occlusion of the medullary tube, abnormal growth of brain tissue, with ultimate formation of cerebral herniæ. A similar explanation, delayed closure of the medullary tube in the occipital region, holds good also for the cases of occipital hernia.

b and c. As to those of the base of the cranium, embryological factors which cannot be discussed here in detail, more or less satisfactorily explain their etiology. In general, one might say that the existence of so-called anterior and posterior neuropori, processus neuroporici, canali cranio-pharyngei and occipital curvature, are sufficient reasons for the occurrence of dystopies, under the influence of trauma, pulling of amniotic adhesions or other external factors which are instrumental in the intrauterine life.

The *clinical picture and course* of cephalomata are variable. In some instances a

cephaloma is firmly adherent to the skin; the walls are thin and tender; the tumor is tense, pulsating synchronously with the radial artery and is quite painful on pressure which causes respiratory troubles. Especially are the above symptoms typical of cephalomata connected with the ventricular cavity (hydrocephalocele).

On the other hand, there are more benign tumors, firm to touch or cyst-like, increasing in size gradually and hardly showing any communication with the contents of the cranium.

Of the general symptoms should be mentioned paralysis, mental deficiency bordering on idiocy, anomalies of the cranium, defective vision, headaches, hydrocephalus, general marasmus. Often there are present other maldevelopmental conditions, such as spina bifida, hare-lip, palatum fissum, etc.

Youth, localization, defective mentality, disturbances of intracranial pressure, anomalies of the skull and the vertebral column differentiate cephalomata from dermoid cysts, atheromata, traumatic brain hernia, etc.

PROGNOSIS AND TREATMENT

The older statistics show that without surgical interference a bearer of cephaloma usually succumbs in early childhood. Thus according to the data of Réali, Schatz, and Miller, out of 244 cases only 9 per cent reach a mature age. If treated surgically, recovery obtains in 50 to 60 per cent (Lissenkoff, Beresniagovsky). Even those that survive the operation are doomed to a miserable existence for they become victims of idiocy, paralysis, blindness, hydrocephalus, optic atrophy, unbearable headaches, separation of the bones of the skull, etc. The foregoing factors render the prognosis exceedingly bad, whether the patient is treated or not. Kehrer doubts whether it is not advisable to let an encephaloma alone.

CAUSES OF POSTOPERATIVE DEATH, INDICATIONS AND CONTRA-INDICATIONS

Causes of postoperative death, as given by various authors, are meningitis, hydrocephalus, postoperative escape of cerebrospinal fluid, destruction of vital portions of the brain and ack of vitality, that is, the patient is rendered incapable of existence. Of the foregoing factors, meningitis is the most frequent cause of death (of 32 cases it occurred in 20), while the continuous escape of the cerebrospinal fluid interferes with the healing of the surgical wounds. Fistulæ are formed with the danger of secondary infection.

As to the question when to operate, we may say that this should be done as soon as possible, for cases not operated upon seldom survive more than a year. An immediate operation is indicated when the tumor grows rapidly, its walls are thinned, or when it is so tense that a rupture is imminent or has taken place, or when signs of marked hydrocephalus are in evidence, accompanied by characteristic clinical phenomena. Some authors (Petroff, Beresniagovsky) think that in the presence of hydrocephalus operation is contra-indicated. It is also contra-indicated when the tumor is solid, the skin is not thinned or damaged, and general symptoms are not present.

In suitable cases, one should operate early. Operations performed during the first week of life give, according to Beresniagovsky, 62 per cent mortality; after the first month, 30 per cent. To avoid sepsis, a plastic occlusion of the cranial defects, according to Lissenkoff's suggesion, is the best method (a bone flap from the supraorbital portion of the frontal bone). Even plastic operations, however, ingenious as they might be do not prevent such complications as meningitis, conjunctivitis, etc. Of the plastic methods the best one for closing the bony or hernial canal in the skull seems to be the use of a periosteal flap from the femur including the subcutaneous tissue. The method of Herzen is of interest. For frontal excephalomata he secures intracranial access to the inner opening of the bony canal and does an autoplastic closure from the inside. Such a method has certain advantages: it prevents not only the protrusion of the stump by actual occlusion of the opening of the osseous canal, but infection as well from the diseased lacrimal passages and eyes. When the transplanted flap is healed the cephaloma may be safely removed. In occipital cephalomata, a periosteal flap alone is sufficient.

To avoid the occurrence of hydrocephalus one should watch for cerebral symptoms and

resort to lumbar puncture which in the hands of Preisich gave excellent results. In cases of hydrocephalocele the wound, after the cephaloma has been resected, should be left open and the stump submerged into the subarachnoid space; if this does not prove successful a radical operation of Anton-Bramann should be resorted to.

CONCLUSIONS

1. Encephalocele is a dystopy of the medullary tube occurring early in embryonic life. It is associated with protrusion of the meninges, brought on by disturbances of lymph and blood circulation and the consequent increased intracranial pressure.

2. Meningocele does not exist as a special morbid condition.

3. The cause of the dystopies of the medullary tube lies in numerous embryological factors, such as processus neuroporicus, neuroporus anterior and posterior, occipital curvature, canalis cranio-pharyngeus and many others, in association with traumatic influences during intra-uterine life.

4. Conservative treatment of encephalocele does not give favorable results.

5. If operation is indicated it should be performed within the first months of life.

6. It consists in radical excision of the tumor and the use of an autoplastic flap (subcutaneous and periosteal) as well as in creation of conditions favorable for sufficient and continuous absorption of the excess of the cerebrospinal fluid and regulation of the intracranial pressure.

I am indebted to Professor Herzen of the first State University of Moscow for valuable advice and suggestions, and to Dr. George B. Hassin, of Chicago, for translating the article.

HYPERCHOLESTEROLÆMIA

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A HYPERCHOLESTEROLÆMIA is one of the two etiological factors in the formation of gall stones. Hypercholesterolæmias are known to accompany certain other conditions of which the most important are atherosclerosis, nephritis, and diabetes. The remarks subsequently made in this communication have reference only to diseased conditions of the liver and biliary apparatus in which these complicating factors are not present and play no part unless otherwise specified in the text.

The actual cause of the disturbance of the cholesterol metabolism leading to a hypercholesterolæmia is not known. The studies of Oertel, to which the views of Stadelmann and Eppinger, Jr. are applicable, show that some unknown activity liberated in the liver cell environment causes specific changes in the cell protoplasm leading, probably, to a decreased production, or, at least, to a lack of proper discharge of bile into the biliary passages and to a resultant retention of bile components in the blood stream. This unknown activity is, undoubtedly, of a chemical nature and can be initiated in one of several ways.

As a physiological phenomenon hypercholesterolæmia is present during pregnancy and persists for some time thereafter in the puerperium. It is entirely possible that a distinct biological purpose is intended, in which case the process may be a constructive one necessary to the growth of the fetal tissues. Such a physiological purpose is most probably accomplished through the agency of some hormone activity derived in the general environment of the pregnancy. Definite knowledge concerning this point is, however, not available at the present writing.

On the other hand, the process may be entirely different. Then it must be assumed that toxic bodies are liberated in the liver cell environment and that they are intimately associated with the increased general metabolism accompanying the growth, development, and life's activity of the fetus. Just which one of the many products of metabolism is at fault, is unknown.

In any case under ordinary circumstances this physiological phenomenon is so controlled as to enable the woman to compensate adequately for the abnormal increase of metabolic effort. It seems reasonable to assume from abundant clinical evidence that in the majority of cases the excess of cholesterol in the body is removed by the natural resources of the liver, and that no abnormality remains which could call forth any manifestations of disease. However, it is highly probable that this unusual accumulation of cholesterol bodies during the early months of the child-bearing period is the cause of the vomiting of pregnancy.

Clinically, a group of cases can be differentiated in which, during the course of a pregnancy or some time thereafter, an attack of right hypochondriac pain, sometimes with vomiting and sometimes with slight grades of jaundice, appears. Two interpretations are possible: In one, the course of affairs after the attack has subsided under appropriate treatment indicates with sufficient probability that the pain is due to changes in the liver environment (increased blood supply, congestion) associated with the handling (absorption, distribution, excretion) of cholesterol and other bodies. The association in a few of the patients of slight grades of jaundice gives a clue to the pain. A greater degree of activity causes a fairly unexpected dilatation of the bile passages and some degree of spasm at the papilla of Vater leading to a relative mechanical obstruction of slight degree. In these cases the metabolic coefficient of compensation is sufficient to overcome the disturbance, and no opportunity is afforded for the precipitation of stones.

In the following case, of which I give the clinical and laboratory notes, the attacks began a short time after pregnancy.

CASE 1. Hosp. No. 155890. The patient, age 38, had had many attacks of right hypochondriac pain; a typical attack of gall-stone colic occurred while patient was in the hospital under observation. At operation a large, distended gall bladder was found which contained no stones, and the walls of which showed no morphological change. A cholecystostomy was done and biliary drainage was instituted. The laboratory facts are shown in Table I.

TABLE I.—LABORATORY FINDING IN CASE I

	Blood cholesterol: mg. per cent	Bile cholesterol: mg. per cent	Urine bile	Stool
Pre-operative July 6	0.290			
Postoperative July 8	.....	0.048	?	colored
9	.....	0.035	?	colored
10	.....	0.028	no	colored
11	.....	0.030	no	colored
12	.....	0.050	no	colored
13	0.156	0.040	no	colored
14	.....	0.042	no	colored

In the second group the course of affairs after the subsidence of the attack indicates that the physiological effort is not sufficient to overcome the extraordinary increase of the cholesterol metabolism and the physicochemical conditions of the bile become such as to favor and cause a precipitation of stones. This was shown exceptionally well in one patient in whom at operation a single stone about 1 inch long and half as broad and thick was found in a gall bladder which otherwise was devoid of any abnormality of structure. The stone was a cholesterol stone, of a soap-like translucency, and with no trace of bile pigment in its structure.

The occurrence of bacterial infection within the liver parenchyma, or in the biliary passages has been found to be associated both with normal and hypercholesterolæmic conditions.

With pure infections limited practically to the gall bladder and, perhaps, to the larger ducts and without obstruction in the terminal part of the duct system, normal blood conditions are always found. Apparently in these cases one is dealing with an infection of the gall bladder which has exact similarities to the ordinary types of infection of the appendix and the element of any metabolic disturbance is an entirely extraneous matter which plays no part in the pathological or clinical picture. Indeed, no stone precipitation need occur. The notes of the following cases illustrate this type of case.

CASE 2. Hosp. No. 161627. A woman, age 36, had had attacks of right sided abdominal colic during the 10 months previous to her admission to the hospital. There had never been any jaundice. Operation showed an empyema of the gall bladder; no stones were present. Prior to operation the blood contained 187.5 milligrams per cent of cholesterol.

CASE 3. Hosp. No. 163056. A woman, age 60, for the past 20 years had complained of upper abdominal pain. For the last 4 days there had been an acute attack, with fever and vomiting and without jaundice. Operation showed a gangrenous gall bladder without stones. Prior to operation the blood contained 157.5 milligrams per cent of cholesterol.

In other cases in which infection is a dominant factor, stones are found in some part of the biliary tract. The notes of the following case illustrate a pure infection of the biliary tract followed by stone precipitation and without disturbance of the cholesterol metabolism.

CASE 4. Hosp. No. 154276. This patient had had typhoid fever when a child. When 50 years of age she was admitted to the hospital with a history of indefinite upper abdominal symptoms which had been present for the preceding 7 years, but which had at no time been associated with nausea and vomiting or with jaundice. An acute attack of cholecystitis occurred twice in the 3 weeks immediately preceding admission to the hospital. Operation showed a thin walled gall bladder distended with a large number of small greenish-black stones. A cholecystostomy was done and the bile was drained through the fistula.

The stones contained practically no cholesterol and were composed of bile pigments. The facts obtained from examination of the blood and bile are shown in Table II.

TABLE II.—LABORATORY FINDINGS IN CASE 4

	Blood cholesterol: mg. per cent	Bile cholesterol			Urine bile	Stool bile
		c.cm	sp. gr.	mg. per cent		
Pre-operative May 5	0.150					
Postoperative May 6	.....	164	1015	0.0625	no	absent
7	.....	80	1010	0.02415	no	absent
11	.....	170	1012	0.02175	no	trace
12	.....	50	1010	0.0250	no	colored
13	0.12625	...	....	.....	...	.....

The great bulk of the cases of cholelithiasis are not so sharply demarcated as the illustra-

tive case just described, and even when infection is a dominant factor at the time of operation, the character of the clinical and laboratory data and of the anatomical findings demonstrated at operation do not always furnish satisfactory criteria for making a judgment. In some of the cases the blood contains normal amounts of cholesterol during the period of observation, as the notes of the following cases illustrate.

CASE 5. Hosp. No. 159292. A man of 36 had his first attack of acute cholecystitis 6 days before admission to the hospital. A remission of the symptoms occurred and was followed by a recrudescence. There was no jaundice. At operation a gangrenous empyema of the gall bladder was found; mixed stones were present. Prior to operation the blood contained 147.5 milligrams per cent of cholesterol.

CASE 6. Hosp. No. 149345. A woman, age 21, had two attacks of acute cholecystitis two weeks and four days before admission. There was no jaundice. At operation an empyema of the gall bladder was found; mixed stones were present. Prior to operation the blood contained 147.5 milligrams of cholesterol per 100 cubic centimeters.

CASE 7. Hosp. No. 159623. This patient had two attacks of cholecystitis 7 weeks and 1 day before admission. There was no jaundice. A gangrenous inflammation was present in the gall-bladder wall; the contained bile was purulent; the gall bladder contained mixed stones. Prior to operation the blood contained 170 milligrams of cholesterol per 100 cubic centimeters.

In other cases of cholelithiasis in which infection is apparently a dominant factor at the time of observation, hypercholesterolæmic conditions are found in the absence of obstruction in the terminal part of the duct system and in the absence of any other complicating factors, such as atherosclerosis, diabetes, and nephritis. The notes of the following cases illustrate this type of case.

CASE 8. Hosp. No. 158117. A woman, age 26, unmarried, had had symptoms for 9 months consisting of right hypochondriac pain and vomiting. Operation disclosed an inflamed gall bladder containing stones of a mixed construction. A cholecystectomy was done. Prior to operation the blood contained 205 milligrams per cent of cholesterol; after operation, 195 milligrams per cent.

CASE 9. Hosp. No. 158405. A woman, age 30, married, had had mild attacks of acute cholecystitis for the 9 months preceding her admission to the hospital. No jaundice was ever present. At operation the gall bladder was found full of pus and stones. A cholecystectomy was done. Prior to operation the blood contained 272.5 milligrams per cent of cholesterol.

CASE 10. Hosp. No. 156562. A patient of 50 years was admitted to the hospital with a history of frequent attacks of gall-bladder colic, with fever and vomiting and without jaundice. At operation the gall bladder was found the seat of a gangrenous inflammation, and it was found to contain a number of mixed stones. Prior to operation the blood contained 212.5 milligrams per cent of cholesterol.

CASE 11. Hosp. No. 158544. A man, age 54, had an attack of acute cholecystitis which continued for the 5 weeks immediately preceding his admission to the hospital. At operation a gangrenous inflammation of the gall bladder was demonstrated and one stone was extracted from the cystic duct. Prior to operation the blood contained 240 milligrams per cent of cholesterol. Thirteen days after operation the blood contained 168.5 milligrams per cent of cholesterol. Twenty-two days after operation the blood contained 492.5 milligrams per cent of cholesterol.

The explanation for these diverse findings with practically similar clinical and pathological pictures is to be found in the biological phenomena of gall-stone formation. In previous studies evidence was presented to show that in clinical medicine both disturbances of metabolism and infection are equally operative in causing a precipitation of stones and that in actual practise no one method occurs to the total exclusion of the other. In certain of the cases infection initiates the pathological process leading to calculus formation. In others, there is a primary disturbance of the cholesterol metabolism. Most commonly this disturbance originates during pregnancy.

For any individual case the available knowledge indicates that thereafter the biological sequence of events includes an infinite variety and number of combinations of infection and disturbed metabolism, which may occur simultaneously, or, more frequently, alternately, and with different degrees of severity. Depending on the factor which is paramount, on the intensity of the latter's manifestations, on the stage of the process, or on the presence or absence of complicating factors, which now become numerous, the pathological picture shows wide variations, and the blood shows complementary wide fluctuations of the amount of cholesterol circulating in its serum. In certain of the cases of hypercholesterolæmia plus infection a vicious circle is

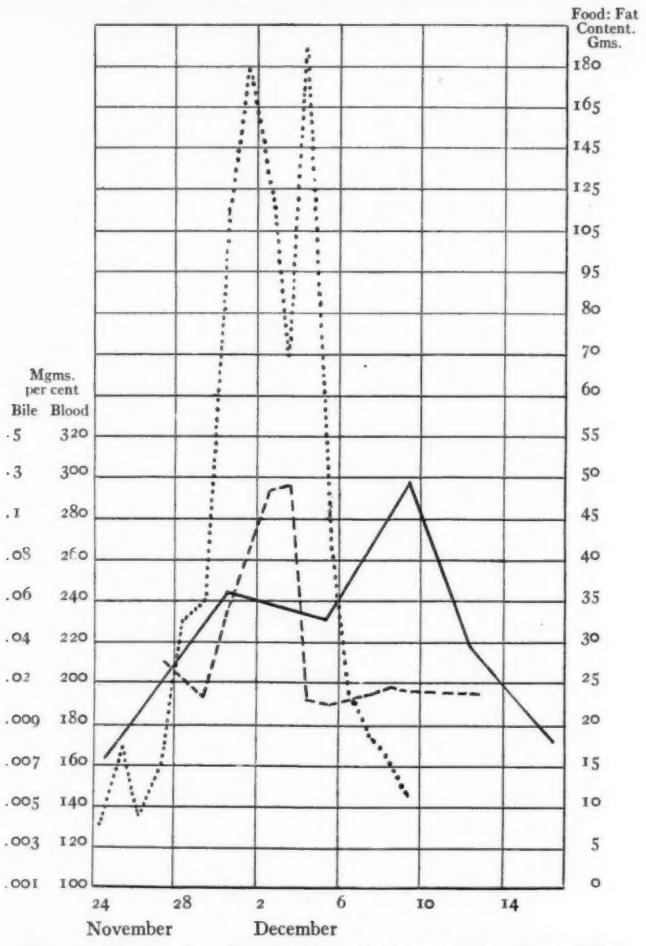

Chart I. Findings in a hypercholesterolæmic patient in whom a biliary fistula had been established for a therapeutic purpose.
Blood cholesterol ———. Bile cholesterol _ _ _ _ _. Food: fat content .....

formed by the two in that the infection by virtue of the action of the toxins thus formed in the liver cell environment interferes with the activity of the liver cell and enhances or continues the retention of cholesterol in the blood.

In such cases of manifest infection associated with gall stones, as are cited in this communication, the presence of a normal cholesterolæmia indicates that at the given moment the cholesterol metabolism is working at par and that no disturbance of its activities exists. This does not, however, give any inkling of the primary factor at fault, nor as to whether at any previous time a hypercholesterolæmia had existed which had spontaneously corrected itself. But it is permissible to assume, whenever the demonstrated stones contain a large excess of cholesterol (75 to 90 per cent), that at some previous time such a disturbance of metabolism had been present.

Under similar conditions—infection plus stones—the presence of a hypercholesterolæmia indicates that some disturbance of metabolism exists at the moment whenever no complicating obstruction is to be found in the terminal part of the duct system. When such obstruction exists, one must give sufficient time for the obstruction to be relieved completely, usually through operative measures, before one can form a judgment as to whether the hypercholesterolæmia is due wholly, or only in part, to the obstruction.

Hypercholesterolæmic conditions have been observed in women in the absence of pregnancy and have also been observed in men, in either case, in the absence of any manifest infection. It must be assumed, therefore, that under such conditions the disturbance of metabolism is produced in other ways and from other sources. The available data give many reasons for believing that under such conditions the disturbance of the cholesterol metabolism resulting in an accumulation and retention in the blood of increased amounts of cholesterol is a fatigue phenomenon. The essential mechanism of the latter occurs in the cholesterol filter, the liver cell. Overactivity of the liver cell, which is usually caused by its having too much product to handle, results automatically in a decrease of cellular activity extending all the way from that of slight diminution to that of total cessation. The best comparison is to be found in the phenomena of fatigue and rest in muscle tissue. Fatigue in muscle is due to an accumulation of certain products of cell metabolism and the degree of fatigue corresponds to the extent of the accumulation and varies from slight grades of "feeling tired" to that excessive grade in which muscular activity gives out completely. Cessation of muscle activity furnishes an opportunity for the elimination of the excess of excrementitious materials and corresponds, clinically, with the disappearance of the state of fatigue and the return of the state of rest. Fatigue in the liver cell is accompanied by a piling up of cholesterol in the blood stream. Opportunity for recuperation is provided by lessening the supply of lipoid delivered to the liver cell, which is accomplished by curtailment of fat in the diet, and by the rapid elimination of bile cholesterol by drainage.

This conception is illustrated in Chart 1, which shows the findings of an experiment carried out in a hypercholesterolæmic patient in whom a biliary fistula had been established for a therapeutic purpose. Originally a hypercholesterolæmic crisis had been present. On November 24, the cholesterol content of the blood and bile had fallen to normal levels under the influence (1) of bile drainage, and (2) of a diet in which minimal amounts of fat were allowed. Thereafter the diet was made to contain very large quantities of fat. Almost immediately the blood contained an increased amount of cholesterol but with the corresponding increase of the output of cholesterol in the bile, the blood content of cholesterol showed a slight tendency to fall. On December 3, the output of cholesterol in the bile reached its highest point and thereafter it fell abruptly to a much lower figure in spite of the fact that the fat content of the food was continued for another 24 hours. This must be interpreted as indicating that at this point the liver cell reached the state of greatest fatigue and thereafter a diminution of its activity occurred. The sudden decrease in the output of cholesterol in the bile was followed by a marked retention of cholesterol in the blood which reached its maximum on December 9. Thereafter the blood content of cholesterol again fell to normal as recuperation was effected and the liver cell reached a state of normal activity.

It is quite possible that in persons with this established diathesis the threshhold to fatigue in the liver cell is abnormally low. This may be due to factors similar to those previously described, that is, to toxins liberated in the liver cell environment. The one aids and abets the other and a vicious circle is thus formed. The place from which these toxins are most likely to originate is the intestinal tract. Such poisonous bodies may, first of all, be derived from the intestinal bacterial flora. Or the toxins may result from the normal digestive processes in which harmful by-products are split off and sent to the liver via the portal system to be neutralized and excreted or otherwise dealt with.

Possibly this conception of fatigue as a cause for the accumulation and retention of cholesterol bodies in the blood furnishes an explanation for the frequently encountered temporary disturbance which passes commonly under the generic term of "biliousness." On purely empirical grounds this has been referred to the liver. Clinically this disturbance is most apt to be met in persons of the florid type who frequently eat a great deal more than they should and are fond of relatively rich foods. Some other associated facts include a thickly coated tongue, a "bad" taste in the mouth, a sluggishness of the bowels with stools that are inclined to be scanty and of much lighter color than is customarily seen, and a general lassitude and inaptitude of all of the body functions, both physical and mental. This corresponds fairly accurately with an insufficiency of liver function; and the alternation of periods in which "biliousness" is or is not present corresponds, in my mind, to alternating periods of fatigue and rest in the liver cell.

In a previous communication reference was made to hypercholesterolæmic conditions which were observed to persist immediately after operation (cholecystectomy, cholecystostomy, either one with and without drainage) and which disappeared only when the bile passages were cleared of one or more stones at a secondary exploration. Other findings included: (1) A sinus leading to one of the larger ducts, the hepatic or common duct. The outer end of the sinus was naturally infected. The presence of infection in the ducts proper could not be determined but from the character of the anatomical appearances of the interior of the duct, as demonstrated at the secondary exploration, it is correct to assume that no inflammatory reaction was present such as one ordinarily sees resulting from a bacterial infection. The absence of chills and fever and of other evidences of a sepsis indicated that there was no infectious cholangeitis within the confines of the liver. (2) The escaping bile was usually of a greenish color, was very turbid, and deposited a heavy sediment on standing.

Inasmuch as preceding the secondary exploration the patient had been deprived of any excess of cholesterol by the continuous discharge of bile from the fistula for a period of several weeks at least, and by curtailing the sources of repletion in the food, it is correct to assume that the hypercholesterolæmia was not due to any abnormally large amount of cholesterol held in the body economy, but that some local factor was causing an interference with the metabolic mechanism whereby a different distribution of what little cholesterol there was present was accomplished; a proportionally larger amount of the latter accumulated in the blood as opposed to that retained in the solid tissues.

The prompt disappearance of the hypercholesterolæmia after the clearing of the ducts of their contained stones, indicates that the latter was the essential cause. The character of the facts here set forth shows that the stones played the part only of a mechanical irritant.

The notes of the following case illustrate the preceding facts. The case has already been published.

CASE 12. No. 172. A woman had been operated upon for cholelithiasis and a cholecystostomy was done. On September 12 there was an attack of

TABLE III.—LABORATORY FINDINGS IN CASE 12

	Temp. Fahr.	Blood cholesterol: mg. per cent	Bile cholesterol			Urine bile	Stool bile
			c.cm.	sp.gr.	mg. %		
Pre-operative							
Sept. 6	99.8	0.155	.....	.....	.....	.....	.....
Post-operative							
Sept. 8	100.	.....	190	1010	0.061	no	no
10	100.4	.....	140	1010	0.037	no	no
11	99.8	.....	190	1010	0.053	no	no
13	103.	.....	130	1010	0.035	no	no
15	100.2	.....	122	.....	0.008	no	no
16	100.2	0.162	270	1008	0.031	no	no
18	99.2	.....	170	.....	0.035	no	no
28	101.4	.....	450	1010	0.031	no	no
29	99.4	.....	530	1010	0.044	trace	no
Pre-operative							
Oct. 1	99.	0.165	.....	.....	.....	no	no
After 2nd operation							
Oct. 14	101.8	.....	111	1012	0.025	no	no
15	101.6	.....	110	1010	0.034	no	sl. col.
17	99.8	.....	.....	.....	0.065	no	colored
21	99.6	.....	.....	.....	0.047	no	well col.
28	99.4	0.125	.....	.....	.....	.....	.....

colic. On September 26, the notes state that the discharge of bile was still profuse, the bile was green and very turbid, and a stone was felt in the gall-bladder sinus which was promptly extracted. Up to October 12, however, the drainage still continued to be just as profuse, and on the following day the bile passages were explored. A secondary cholecystectomy was done and a stone was removed from the papilla of Vater. The character of the drainage changed immediately and the color of the bile became clear golden yellow. The convalescence was uneventful thereafter. The laboratory facts are given in Table III.

In another communication reference was made to the disturbances of the cholesterol metabolism which occurred at later periods and sometime after operations for gall stones and gall-bladder disease were apparently successfully carried out. A number of reports (Eisendrath, Deaver, Judd and Harrington, Deaver and Reimann, and Davis) were mentioned which fully describe the cases in which postoperative symptoms have been sufficient to require secondary operations, the actual cause being fistula, stone, cholangeitis or obstructive jaundice. Such factors have only an indirect and contributory value in any accompanying disturbance of the cholesterol metabolism except in those stone cases in which it can be definitely proved that the calculi, which are subsequently found in the ducts, were formed since the primary operation.

The usual opinion that the stones which are found at secondary operations are the results, possibly beyond the control of the operator, of inefficiently done primary operations in which all of the stones are not removed, is an assumption which seems to be entirely correct in the majority of the cases.

In such cases of postoperative common and hepatic duct stone, which we have studied, there has always been present a hypercholesterolæmia. Jaundice has usually, but not always been present. There was no acute inflammation (infectious cholangeitis) of the bile ducts present at the time of the secondary exploration. Naturally the secondary choledochotomy was followed by a profuse discharge of bile. The escaping bile was clear or, at most, contained shreds of inspissated mucus and the latter, when present, disappeared completely in the first few hours of drainage. Following the secondary operation in which the ducts were explored and the stones removed, there was a very prompt disappearance of the hypercholesterolæmia.

In the presence of a discharging biliary sinus and in the absence of any jaundice, the mechanism is similar to that described in the preceding paragraphs. In the presence of jaundice a relative obstruction is present especially in the capillary bile ducts which is due to swelling of the lining cells.

Duct stones in the presence of a healed wound and in the absence of jaundice have also been observed with hypercholesterolæmia. The stone acts as a foreign body and causes an interference with liver cell activity leading to a diminution of function. In some of the cases, undoubtedly, there is also primary metabolic disturbances along the lines previously indicated. Except when definite knowledge is had from previous observation and study of the behavior of the cholesterol metabolism, a final judgment is frequently not possible until some time has elapsed after operation and the postoperative course has been adequately observed.

If, with a healed wound and a stone in the large ducts, an attack of colic supervenes, an obstruction forms, more or less temporarily, at the point where the stone becomes impacted. Back pressure is exercised upon the duct system and upon the liver cells which interferes with cell activity. In such cases one must give sufficient opportunity for the obstruction to be completely and sufficiently relieved by operation and by thorough clearing of the ducts before one can make any judgment as to whether the hypercholesterolæmia is due in whole or in part to the obstruction.

Somewhat analogous facts are to be found with stones and obstructions in the urinary tract. A stone in any portion of the ureter or pelvis causes an interference with the functional activity of the kidney as shown by the various dye tests. Obstructions at the urinary outlet, the prostatic urethra, also cause diminution of kidney function. In these respects the analogy between liver and kidney function is very strong.

It is conceded by nearly everyone that stones do occasionally form and reform in the ducts after cholecystectomy. And cases are

well known, if rather uncommon, in which intrahepatic stones are formed high up in the confines of the liver from which they are being discharged continuously. As a part of this clinical picture a chronic cholangeitis is presupposed.

In the cases which we have studied, a moderate or well marked cholangeitis of this type accompanied with intrahepatic stone precipitation has been rare. In one of the cases the cholangeitis was associated with a continuous but variable grade of jaundice in the absence of any demonstrable hypercholesterolæmia. The stones which were passed from the biliary (common duct) fistula were very small. They were, for the most part, mere granules of a dark greenish-black color, and contained very little, if any, cholesterol. In another patient, an intrahepatic precipitation of stones occurred and became continuous; a hypercholesterolæmia was present; the stones, however, were composed mostly of biliary pigments and to a less extent of cholesterol. In both of these cases the biliary fistula showed little tendency to close; the amount of biliary discharge was usually of moderate amount, although there were times when for a number of days it became of larger quantity. It was possible to cultivate bacteria from the biliary discharge. Bile was present in the stools in diminished quantity, except at rather rare intervals; then apparently some obstruction took place at the terminal part of the duct system and the stools become clay colored.

It is known that as time goes on a certain amount of connective tissue change is bound to take place around the intrahepatic bile passages and a form of biliary cirrhosis results. The occurrence of the latter, probably, has some influence upon the cholesterol metabolism.

Up to the present time the experience with cases of true cholangeitis with and without intrahepatic stone precipitation has been extremely small and the subsequent discussion is, therefore, given with a certain amount of reservation; a final opinion is not possible at the present writing. However, previous studies have shown that the jaundice accompanying cirrhotic liver changes is unaccompanied by any increase in the cholesterol content of the blood. This finding is a constant one. This observation furnishes a satisfactory explanation of the discrepancy in the cholesterol contents of the blood of the two cases described in the preceding paragraph; in the first a biliary cirrhosis was probably present; in the second, it probably was not.

The occurrence of a hypercholesterolæmia with intrahepatic stones of cholangeitic origin seems to depend on a number of phenomena. It is usually noted that the total amount of bile secreted by the liver is not up to the normal. This is probably due (1) to the swelling of the walls of the bile passages with a consequent narrowing tending to impede the outflow; and (2) to the swelling of the liver cells themselves accompanying the marked congestion of the liver parenchyma. The views of Stadelmann and Eppinger, Jr., which were referred to previously, are especially applicable, namely, that toxic substances are liberated in the liver cell environment which cause specific changes in the liver cell environment and which lead to a decreased production, or, at least, to a lack of proper discharge of bile into the capillary bile passages and to a consequent retention of bile components in the blood stream. In any case it seems reasonable to assume, at present, that the underlying factor is a chronic infection of the intrahepatic bile passages, that the concretions have an infectious origin, that any hypercholesterolæmia, which is demonstrable, is due to duct obstruction and interference with liver cell activity and the consequent retention of cholesterol bodies in the blood stream, and that there is no underlying derangement of the cholesterol metabolism leading to an increased production of lipoid bodies except as they form consequences of the toxic processes causing an interference with cellular activity.

ADHESIONS ABOUT THE ASCENDING COLON SIMULATING CHRONIC APPENDICITIS[1]

By CHARLES DAVISON, A.M., M.D., F.A.C.S., MARSHALL DAVISON, B.S., M.D., and DON J. ROYER, M.D., Chicago

WITHIN the past 2 years our attention has been drawn to a group of cases which seem to present a new and definite surgical entity. They are of particular interest because of the ease with which the condition may pass unrecognized with a consequential non-relief of the subjective symptoms of the patient, and a reflection on the ability of the surgeon.

The interest in this group of cases was initiated by our observation of a number of patients who presented a recurrence or non-relief of symptoms following operation for acute or chronic appendicitis. Closer observation of cases giving symptoms of chronic appendicitis, in combination with roentgenographic examination, has led us to the conclusion that the condition is not a rarity, but is of a comparatively frequent occurrence.

The symptoms are those of a vague abdominal condition, and are alike in those that have been operated upon for appendicitis with no relief, and in those who present themselves with no history of previous surgical treatment. In some instances the patients have been treated medically for a chronic gastro-intestinal disorder, such as peptic ulcer, colitis, chronic constipation, or gall-tract disease. The relief under such management has been either temporary or absent, with a more or less rapid return of symptoms after a period of improvement.

The disassociation of such a syndrome with any definite condition led us to resort to complete gastro-intestinal examinations by means of the barium meal and X-ray in an effort to make a definite diagnosis, and it was by this means that the condition was first brought out. Such examinations revealed a definite and more or less pathognomonic disfiguration of the shadow contour of the ascending and transverse colon, which seemed to be caused by bands of adhesions passing over the ascending colon, involving to various degrees the transverse colon, and producing a partial or complete obstruction of the large bowel at the point of greatest involvement. In most instances the roentgenogram revealed a ptosis of the transverse colon, an agglutination of it to the ascending bowel, with a consequent kinking or constriction at the hepatic flexure. These roentgenographic findings are definite, and laparotomy reveals the mechanical condition exactly as it is shown by the plates and fluoroscope. So absolute are these findings that we are able to diagnose definitely before abdominal section the exact position and extent of the pathology. With operative interference and a mechanical correction of the pathology present, the subjective symptoms are quickly relieved, and to date we have had no recurrence reported.

A brief résumé of a few typical cases, with reference to the roentgenograms, will aid materially in the interpretation of a more detailed discussion. In each instance, only a short outline of the case is given.

Case 1. B. W., male, Jewish, age 36, clothier, married. For the past 3 weeks the patient has complained of a dull, aching pain extending from the right kidney region downward into the lower right quadrant of the abdomen. It has not been sharp or cramping at any time, but has been severe enough to cause nausea. He has been troubled with digestive disturbances of an indefinite nature for several years. He is chronically constipated, and at times is bothered a great deal by gas. The patient states that his appendix was removed 17 years ago.

Physical findings. A generalized abdominal distention of moderate degree is present. There is marked tenderness and rigidity over the entire right half of the abdomen. No definite palpable mass is present, but there is a feeling of different resistance to this portion. Leucocyte count 16,000. Urinalysis negative.

Roentgenographic findings. (Fig. 1.) Examination by barium meal. The stomach and small bowel showed no pathology. At the twenty-four hour observation, there was evidence of constrictions at the junction of the cæcum and ascending colon, and the transverse colon was adherent to the ascending portion. This remained the same at the forty-eight

[1] Read before the Chicago Surgical Society, April 6, 1923. (For discussion, see p. 277.)

hour observation despite catharsis and numerous enemata, which were given with the intention of freeing the bowel of the opaque media, so that the right kidney might be visualized.

Operative findings. A high right rectus incision was made. The cæcum and ascending colon were markedly distended with gas and fluid, and somewhat discolored. The old appendectomy scar was covered with omentum. At the upper portion of the ascending colon there crossed a thick, fibrous band of adhesions, fastening themselves to the transverse colon just beyond the hepatic flexure, and producing a sharp angulation at that point. There was not a marked degree of ptosis of the transverse colon, and with the exception of the point of attachment of the band, there was no agglutination of the two portions of the colon. The band of adhesions was dissected off, a small portion of the denuded gut peritonized, and the abdomen closed.

CASE 2. N. P., male, Italian, age 56, laborer, married. The patient has been bothered with constipation for years, which has been steadily growing worse the past 6 months, until now the bowels will hardly move even with the use of cathartics. At times he notices a tumor mass in the right lower quadrant of the abdomen. This mass is about the size of a man's fist, slightly tender, seems to move about the abdomen, and disappears after defæcation. There is a severe cramping pain in the abdomen during the attacks of constipation, which is relieved by defæcation and the expulsion of gas. There is left, however, a dull, constant pain, which remains until the next attack. He is nauseated at times, but has never vomited. Appendix removed 4 years ago.

Physical findings. There is no noticeable difference in the contour of the abdomen. A moderate degree of tenderness and rigidity is present over the ascending colon and hepatic flexure, with slight tenderness and rigidity over the entire abdomen. There are no tumor masses palpable. Leucocyte count 6,200. Urinalysis is negative.

Roentgenographic findings. Examination by barium meal. The stomach and small bowel showed no evidence of pathology. At the twenty-four hour observation the ascending and transverse colons were found to be adherent, and there were apparent constrictions at the hepatic flexure and the middle third of the transverse colon (Fig. 2).

Operative findings. A right rectus incision was made at the upper quadrant. The cæcum was drawn up and inspected, and found to be dilated but freely movable. The transverse colon was ptosed, rotated anteriorly and bound to the ascending colon about 6 centimeters below the hepatic flexure by a band of the same width arising from the lateral peritoneal wall. The adhesions were freed, and the gut peritonized.

CASE 3. S. F., male, Italian, age 55, retired, married. For the past 6 months the patient has had pain in the entire right abdomen and stomach. The pain extends from the ribs downward, is acute in type, with nausea and vomiting, and seems to be slightly relieved by bowel movements. The patient has had stomach trouble for years, which he thinks has been due to constipation. His appendix was removed 5 years ago.

Physical findings. There is tenderness over the entire right side of the abdomen, most marked at the upper quadrant. Slight rigidity is present, but no palpable masses. Leucocyte count 10,000. The urinalysis is negative.

Roentgenographic findings. Examination by barium meal. The stomach showed delayed emptying time. There is evidence of a pathological gall bladder, with periduodenal adhesions. At the twenty-four hour observation, the cæcum was found to be bound down in the lower right quardrant, and the ascending and transverse colons were adherent. This condition remained constant over a period of seventy-two hours, after which the examination was discontinued (Fig. 3).

Operative findings. An incision was made over the right rectus muscle in the upper quadrant. Adhesions were found binding the transverse colon to the liver and surrounding the gall bladder. The pyloris was also bound to the under surface of the liver. The ascending colon and cæcum were bound down by bands of adhesions arising from the lateral parietal wall. The gall bladder was markedly thickened, and slightly enlarged. The adhesions about the gall bladder, pylorus, and liver were broken up; the cæcum and ascending colon were freed and peritonized, and the gall bladder was drained.

The patient died on the third day from a paralytic ileus.

CASE 4. P. B., female, American, age 30, stenographer, single. The symptoms began a short time after an appendectomy 2 years ago. The patient complains of pain in the back radiating to the right lower quadrant of the abdomen. This has been noticeable for the past year, but has become worse in the last 4 months. At times the pain radiates to the right hip and leg. It is of a dull, constant, aching character, with no acute attacks. Headaches are frequent. Nausea, but no vomiting, is present for 2 or 3 days before menstruation. Appetite is poor. The patient is always constipated.

Physical findings. There is tenderness at point of old right rectus scar extending upward to the gall bladder region. There is no tenderness in the back. Leucocytosis 9,600. Urine is negative.

Roentgenographic findings. Examination by means of a barium clysma. The colon was infiltrated and appeared normal to about the middle third of the transverse portion, where there was an apparent constriction. There was difficulty in getting the barium through the hepatic flexure and into the cæcum. The cæcum was bound down below the crest level, and the ascending and transverse colons were adherent to each other (Fig. 4).

Operative findings. An incision was made at the region of the old scar. The omentum was adherent to the anterior parietal peritoneum at this point. There was an adhesive band about 4 inches in width

extending from the parietal peritoneum over the cæcum and ascending colon up to the proximal third of the transverse colon. There was ptosis and anterior rotation of the transverse colon, with angulation at the hepatic flexure. The cæcum and ascending bowel were markedly dilated. The adhesions were broken up, the colon freed, and peritonized.

CASE 5. J. G., male, American, age 60, banker, married. The symptoms have been present for an indefinite time. A dull pain is present in the right side, which steadily has been growing more noticeable for the past year. The pain never was severe until 3 months ago, when it became more intermittent and of a cramp-like nature. The patient has had indefinite digestive disturbances with frequent eructations of gas after eating. He never has a bowel movement without the use of a mild laxative. A short time ago he was given the diagnosis of chronic appendicitis by his physician.

Physical findings. The contour of the abdomen is normal. There is slight tenderness and rigidity over the region of the appendix, which extends to the upper quadrant of the abdomen. Leucocyte count 14,000. The urinalysis is negative.

Roentgenographic findings. Examination by means of a barium meal. The stomach and small bowel showed no evidence of pathology. At the twenty-four hour period there was evidence of a pathological retrocæcal appendix, and there was definite evidence of a circular adhesion constricting the large bowel at the junction of the cæcum and ascending colon (Fig. 5).

Operative findings. A right rectus incision was made. A partially obliterated appendix was found lying retrocæcally bound against the cæcum by a mass of adhesions. The appendix was dissected out and amputated. The adhesions also bound the ascending colon to the proximal portion of the transverse colon, producing a constriction of the lower portion of the ascending bowel. There was no obstruction at the hepatic flexure. The gall bladder and duodenum were negative.

CASE 6. B. E., female, Jewish, age 20, stenographer, single. Indefinite gastric distress and vomiting have been present for the past 6 months. There have been no acute attacks, but the patient has been bothered by a dull pain and discomfort over the entire right side of the abdomen. Vomiting occurs at infrequent intervals, and seemingly has no relation to the pain. The patient is habitually constipated. Dull headaches are present every few days. There has been a slight loss in weight.

Physical findings. There is marked rigidity and tenderness over the entire right side of the abdomen, especially marked over the region of the appendix. White count 11,000. Urinalysis negative.

Roentgenographic findings. Examination by means of a barium meal. The stomach showed no evidence of pathology. There was a reflex condition in the duodenum causing a gastric stasis. At the twenty-four hour observation there was evidence of a pathological retrocæcal appendix with paracæcal adhesions. The ascending and transverse colons were adherent, and there was evidence of spastic colitis (Fig. 6).

Operative findings. A high right rectus incision was made. A long injected appendix, not involved in any adhesions, was removed. The gall bladder, tubes, and ovaries were normal. A small band of adhesions arising from the lateral parietal wall extended over the ascending colon, producing a slight constriction, and binding down the region of the transverse colon just distal to the hepatic flexure, producing a moderate degree of kinking at that angle. The band of adhesions was cut and the bowel liberated and peritonized.

CASE 7. A. G., female, American, age 47, housewife, widow. The symptoms have been present for 3 years. The patient complains of pain in the right side of the abdomen radiating to the back. This pain is described as being of a pulling, dragging nature, and worse after doing heavy work. It is not influenced by the taking of food. When the pain is severe it is accompanied by nausea, but the patient does not vomit. She has been habitually constipated for years.

Physical findings. There is marked tenderness and rigidity over the entire right side of the abdomen, with exquisite pain in the right lower quadrant. There is slight abdominal distention, and a gurgling sound may be heard on auscultation. There are no palpable masses. Leucocyte count 7,200. Urinalysis is negative.

Roentgenographic findings. A barium meal was given. There was evidence of an old duodenal ulcer. At the twenty-four hour observation the appendix was not noted. There were adhesions between the ascending and transverse colons, along the entire length of the former (Fig. 7). This condition was still present at the forty-eight hour observation.

Operative findings. On abdominal section the transverse colon was found bound to the entire extent of the ascending colon by a broad band of fibrous adhesions covering over both portions of bowel. This was freed and the two portions of bowel liberated and separated. The gall bladder was atrophied to the size of a hazel nut, and contained no stones. A Riedel's lobe of the liver was present. A long fibroid appendix, lying retrocæcally with no adhesions, was removed.

CASE 8. G. W., male, American, age 44, foreman, married. The symptoms have been present for 10 years. He compains of pain at irregular intervals which is of a dull, constant nature, located in the epigastrium, right hypochondrium and right lower quadrant. The patient has had three attacks of an acute abdominal crisis which passed off in a few hours. Constipation is always present and the pain is worse when this is the most marked. He also is troubled with eructations of gas after eating. He is sometimes nauseated, but has never vomited except during an acute attack. His appetite is poor.

Physical findings. There is marked tenderness and rigidity over the right side of the abdomen from

the hypochondriac to iliac regions. Tenderness in epigastrium is present, but not marked. There is a moderate amount of abdominal distention, and the abdomen is tympanitic with the exception of the right lower quadrant, where resonance is impaired. White count 6,400. The urine showed no findings.

Roentgenographic findings. A barium meal was given. There is definite evidence of a duodenal ulcer. (This perforated the evening of the first day of the examination.) At the twenty-four hour period the cæcum was found to be down below the crest level. The ascending colon and transverse colon were firmly adherent (Fig. 8). This condition was still present at the forty-eight hour period. The patient returned for operation 5 days later, and fluoroscopy at that time showed practically the same condition to be present.

Operative findings. (Case operated by Dr. Karl Meyer.) On opening the abdomen a perforated gastric ulcer was found. There was a plastic exudate between the stomach, liver and intestines, with free fluid present. The perforation was closed and peritonized. A dense band of adhesions about 6 centimeters in width covered the ascending colon, and attached itself to the transverse colon just beyond the hepatic flexure, bringing the two portions of bowel into apposition. This band was cut and dissected from the bowel wall, but peritonization of the denuded portions was impossible. A long retrocæcal appendix was found bound in adhesions which were distinct from those composing the band found at the hepatic flexure. Appendectomy was performed, and the abdomen closed without drainage.

CASE 9. J. K., male, Lithuanian, age 42, machinist, married. Duration of symptoms given as one year. The patient complains of gaseous eructations, constipation, and abdominal pain. The pain is located in the right hypochondrium and epigastrium, and is worse after eating. It is dull and aching in character, never acute, and sometimes radiates to the back. The patient is at times nauseated, but never vomits. He has been treated medically for duodenal ulcer, with no relief.

Physical findings. The abdomen is scaphoid in contour. There is slight tenderness over both upper and lower quadrants of right side of abdomen, but no rigidity is present. Otherwise the findings are negative. Leucocyte count 10,000. The urinalysis is negative.

Roentgenographic findings. Examination of the stomach by means of a barium meal revealed no pathology. The duodenum showed evidence of an ulcer. At the twenty-four hour observation, the appendix appeared retrocæcal and pathological. There were definite adhesions between the ascending and transverse colons (Fig. 9).

Operative findings. A high right rectus incision was made. The ascending colon was found bound to the transverse colon at the region of the hepatic flexure by a band 4 centimeters wide, arising from the lateral peritoneal wall. The ascending colon was dilated and filled with fæcal matter. A chronic retrocæcal appendix also was found, but with no adhesions about it. The adhesive band was cut, the bowel peritonized, and the appendix removed.

CASE 10. M. R., male, Jewish, age 35, salesman, married. The following symptoms have been present for 2 years. Pain in the right side radiating from the lower quadrant upward to the costal arch. A feeling of discomfort in the abdomen is always present, with periods of indigestion at long intervals. The patient vomits occasionally. He has had three acute attacks within the past 2 years which were diagnosed as being either appendicitis or cholecystitis. The patient is habitually constipated.

Physical findings. There is tenderness in the right side of the abdomen from the lower to upper quadrants, with some accompanying rigidity. There are no palpable masses. No distention is present. Tenderness over the gall-bladder area is no more marked than in other regions of the right half of the abdomen. Leucocyte count 15,400. The urinalysis is negative.

Roentgenograms of this case were not made.

Operative findings. There were bands across the colon from the lateral parietal peritoneum to the proximal third of the transverse colon. This was drawn down against the ascending bowel, and a marked angle was thus made at the hepatic flexure. The gall bladder and region of the duodenum were negative for pathology. A chronic obliterated appendix was found and removed. It was free from any adhesions whatever. The adhesive band was cut, and the two portions of the bowel separated. Only slight peritonization was necessary.

To us the etiology of this condition still is somewhat vague and not definitely determined. That it is not a congenital condition is proven by the facts that it is not evident at birth, that it is more frequent later in life, and that it seems to be a progressive affair. An analysis of the age of the patients affected leads only to the conclusion that its occurrence is at the time when all chronic abdominal conditions are most frequent, i.e., from middle adult life upward. Sex would seem to have no influence, as in this series the occurrence is about evenly divided. It is of interest to note, however, that all of the patients have led sedentary lives, and all give a history of chronic constipation. This probably is the most important single factor in the entire etiological chain.

With such an assumption we naturally turn to the theory of infection. Chronic constipation is but another term for intestinal stasis, and such stasis necessarily must be at its greatest degree in the large bowel. As a reac-

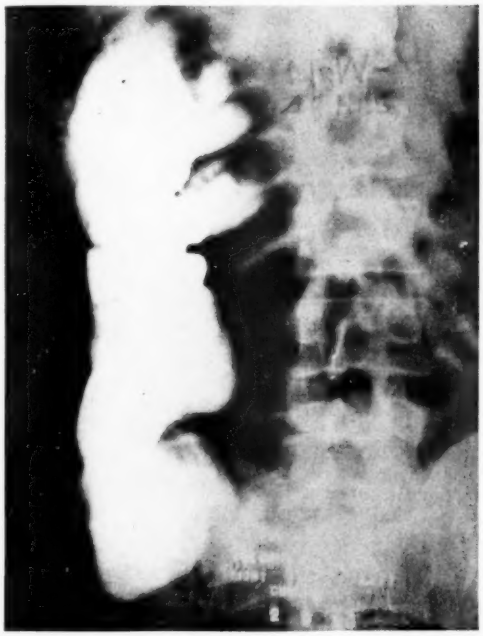

Fig. 1. Case 1. Roentgenogram 48 hours after barium meal, showing practically complete obstruction at hepatic flexure. The ascending and transverse colons are bound together by adhesive band at the hepatic flexure. Gas is seen in the ptosed transverse colon.

Fig. 2. Case 2. Roentgenogram 26 hours after barium meal. Shows agglutination between ascending and transverse colons by circular band at hepatic flexure extending over to middle third of transverse colon, and producing marked ptosis.

tion to the constant accumulation of fæces and resulting atonicity of the bowel, there naturally results a lowered resistance of the gut wall, and an opportunity for the migration of bacteria through to the peritoneal surface and a consequent inflammation. That the infection must be low grade is obvious, otherwise it would result in a more diffuse and acute form of peritonitis. The adhesions which would result from such an inflammatory change seem to occur locally at the point of greatest stasis, i.e., at the ascending colon. We know that for this reason the dependent location of the appendix makes it a constant cess pool of infection, and that when its ability to empty is lost, then occurs an acute or chronic appendicitis depending upon the virility of the organisms.

There is no doubt but that a chronic appendicitis is often found accompanying the condition. However the appendix is not always implicated in the adhesive bands, and very often is free, while a higher portion of the gut will show the distinct pathology. In this regard, four of the series of cases have had an appendectomy performed at intervals from 2 to 17 years before coming under observation for a new condition. This may mean one of two things: either the condition was present at the time of operation and escaped notice, or had its inception and progression at a later date. In the first instance, removal of the appendix evidently had no effect on the growth of the adhesions, or we would have had a cessation of symptoms instead of a condition growing progressively more marked. The second instance again would seem to prove that the presence of the appendix would not necessarily be a determining factor in the production of such regional adhesions. A chronic appendicitis likely has the same etiology as has the production of these bands, but it is an accompanying condition, and not a cause.

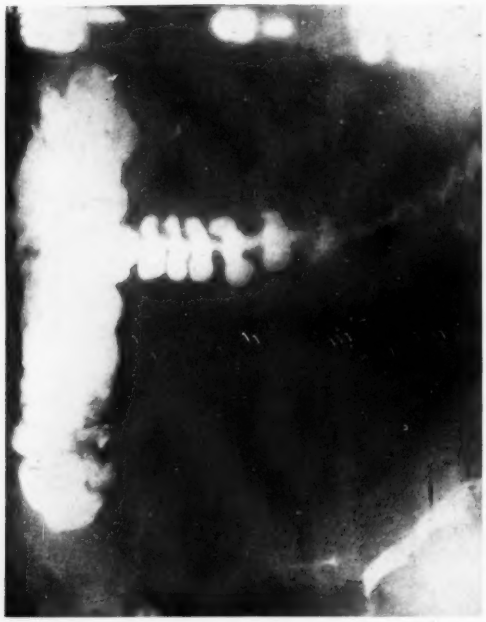

Fig. 3. Case 3. Observation at 48 hours showing complete fixation of proximal portion of transverse colon to area of ascending colon at hepatic flexure by band extending across upper portion of ascending colon. Notice the marked spasticity of the free portion of the transverse colon, which is found in practically all cases.

Fig. 4. Case 4. Roentgenogram after barium enema under compression, showing marked ptosis of transverse colon with traction angulation at the hepatic flexure, and agglutination of transverse to upper portion of ascending colon. The obstruction is incomplete. Outline of gas shows distention of ascending colon.

The fact that these bands seem to have a tendency to involve the colon at the region of the hepatic flexure naturally would lead us to look for such inflammatory pathology in that region as a chronic gall-tract disease, or a duodenal or pyloric ulcer. In only one case (Case 3) was any pathology found in the upper right quadrant which might have been an exciting cause, and in this instance the adhesions about the gall bladder were distinct, separated, and of an entirely different type from those that came upward from the ascending colon.

Ptosis of the transverse colon would be a very important determining factor. It will be noticed that in practically every case this condition has been found to be present, but it is no doubt more marked after the transverse colon has been dragged farther downward by the influence and retraction of the bands. If it existed early only to a slight degree, however, it would mean a definite angulation at the hepatic flexure, with a consequent slowing of the fæcal current over that portion and a resultant stasis in the ascending colon of a more marked degree.

So, it seems to us that the determining factor in the production of this condition is the stasis in the ascending colon, with the resultant possibility of band formation at the point of lowest resistance of the bowel wall.

Pathologically, the condition seems to occur in two forms. In the first, and most frequent (Fig. 10) the band seems to arise from the junction of ascending colon and lateral parietal peritoneum, spreads in a rather fan-shaped manner over the ascending colon, upward and onto the transverse colon, attaching to it at a short distance from the hepatic flexure. Evidently by contraction of the band the transverse colon is rotated anteriorly and further ptosed, bringing it into a variable degree of apposition with the ascending bowel, and thus producing a marked angulation at the hepatic flexure, and a partial obstruction.

Fig. 5. Case 5. Observation after 30 hours, showing partial obstruction at hepatic flexure, slight ptosis of transverse colon, and its approximation to the ascending colon by traction.

Fig. 6. Case 6. Showing partial obstruction of barium at 24 hours. Transverse colon is acutely angulated to lie anteriorly to ascending portion by traction of band in region of hepatic flexure. Gas is seen in ascending colon.

The second type (Fig. 11) may be but an earlier degree of the condition just described. In this type, the band does not reach across to the transverse colon, but rather seems only to involve the ascending portion. Evidently there occurs a thickening or contraction of the band which diminishes to a variable degree the lumen of the colon, with again a chronic obstruction. In either case they seem to arise at points varying from the mid-portion of the cæcum to a position about two-thirds the height of the ascending colon. In no case were they limited to an involvement of the cæcum, but appear to have a tendency to originate slightly higher, and to spread upward, figuratively, as if reaching for a portion of the transverse colon. In width we have seen them vary from 2 centimeters to a size sufficient to cover practically the entire upper two-thirds of the ascending colon and the proximal half of a ptosed transverse bowel, making the two portions of the colon appear at first glance as if one.

But these differences are matters of location, extent, and mechanical effects. In gross histological appearance the bands are identically the same. They are of the dull, glistening, white color of fibrous tissue, varying in thickness, but always too dense to be called a membrane. They are highly vascularized with many small vessels which seem to arise and spread out from the point of origin of the bands. In some instances the bowel wall may

Fig. 7. Case 7. Taken 24 hours after barium meal, showing transverse colon markedly ptosed and bound to entire length of ascending colon. Under the fluoroscope, it was not possible to separate the two portions of the bowel.

Fig. 8. Case 8. Thirty hour barium observation showing transverse colon bound to ascending colon by adhesive band, producing obstruction at hepatic flexure. This roentgenogram was practically unchanged five days later.

Fig. 9. Case 9. Observation at 24 hours showing fixation of transverse colon to upper half of ascending colon by band passing over the ascending colon. Notice the acute angulation at the hepatic flexure.

be visualized through this overlying structure, but in others the density is too great. The surface has almost the smooth glisten of normal peritoneum, but close inspection will reveal a definite limitation of the new tissue and its difference from the true peritoneal appearance.

Although on dissection there is a definite plane of demarcation between the peritoneal coat of the bowel and the under surface of the band, the agglutination between the two surfaces is so close that it is impossible to separate the two without deperitonizing the gut wall. Removal of the adhesive bands leaves a raw, bleeding surface, and has the same effect on the under surface of the removed tissue. Because of the close agglutination between the two, a sliding motion of the bowel in its peristaltic movements beneath the band is not possible, and any manipulation of the intestine means a movement of the entire mass to the limits of attachment of the adhesion. This condition is definitely not analogous to the membrane described by Jackson. It is not a *membrane*, but a definite *band;* it is not limited to the cæcum, and in fact does not seem to involve it to any great degree. Furthermore, it is closely attached to the peritoneal coat of the bowel from which it is impossible to be separated. Jackson's membrane is a veil-like growth, thin, transparent and vascular, which spreads over the cæcum. It is not attached to the covering of the bowel; movements between the two are possible, and careful removal of the veil does not injure the peritoneum of the gut wall.

As has been said earlier, a pathological appendix is often found with the condition. But that the two go together as accompanying pathological entities, both results of the same cause and one not of the other, seems to be proven by the fact that when a chronically inflamed appendix is found, even in the presence of adhesions, the two conditions seem always to be definitely separated and to have different grades of pathology. Again, there is that group of cases in which there are no adhesions about either appendix or cæcum, and, finally, the group associated with a normal appearing cæcum and appendiceal scar.

The symptoms of this condition simulate

Fig. 10. Anatomical diagram illustrating one phase of the pathology. The band has its origin at junction of colon and lateral parietal peritoneum, passes over the ascending colon and is attached to the transverse portion. There results a traction ptosis and anterior rotation of the transverse colon, and fixation of it to the approximated ascending bowel.

Fig. 11. Diagram illustrating different type of same pathology. In this the band does not reach to affect the transverse colon, but has its entire constricting influence on the ascending portion. Notice slight degree of ptosis of the transverse colon.

the ordinary syndromes of chronic abdominal conditions, especially that of chronic appendicitis. The chief complaint has been of a vague abdominal pain, in most cases located in the right side of the abdomen, but not definitely limited to the region of the right lower quadrant, as it generally is in a definite chronic appendix. The area of pain has a tendency to spread upward to involve the region of the right hypochondrium, and often radiates to the back. There is also an indefinite history of digestive disturbances, such as a feeling of heaviness after eating, anorexia, gaseous eructations, nausea and occasional vomiting. Constipation is the rule, and is generally so chronic that it is put in a minor quantity by the patient. Acute abdominal crises arise in this condition in instances in which there occurs a complete or almost complete mechanical intestinal obstruction, and in those instances the symptoms are classical.

The physcial findings are slightly more definite than are the symptoms. The abdominal tenderness and rigidity are somewhat more diffuse than we find them in cases of chronic appendicitis, and extend higher toward the costal arch, without involving, however, the painful area generally found in gall-bladder disease. There is no one exquisite point of tenderness, but it is diffuse over the entire region of the ascending colon to the hepatic flexure.

The leucocyte average in our series of cases was 10,000, with a minimum count of 6,200 and a maximum of 15,400. The variation may be explained on the basis of differing individual reactions to infection, amount of intestinal obstruction and consequent toxic absorption, and the condition of the appendix when present.

It may be seen from the above discussion that such a train of symptoms simulating a chronic appendicitis would be of value only in those cases in which the appendix previously had been removed, and in such instances they prove to be definite of the condition described. In any instance they may simulate any one of several chronic abdominal conditions, and in order to arrive at a definite differential diagnosis, we resort to the use of roentgenographic examinations. By this means we are able not only to rule out the presence or absence of other conditions along the gastro-intestinal tract, but we are able to remove any doubt as to the suspected condition. Too much dependence on symptoms and physical findings often will lead to an incorrect or incomplete diagnosis. We do not believe dogmatically in diagnoses which have for their basis laboratory findings with too little regard for symptoms and physical examinations. But here we have a condition which can be absolutely and definitely diagnosed by the roentgenographic findings and a complete diagnosis is possible in no other way.

In the roentgenographic examination of these patients we prefer the barium meal. The clysma rarely fails to locate the pathology, but has for its disadvantage the fact that we may fail to discover pathology higher up in the gastro-intestinal tract. The cases are examined by both plate and fluoroscope, at frequent intervals immediately after ingestion of the meal and then at successive intervals of 6, 8, and 12 hours. The observations taken at 24, 36, 48, and if necessary, 72 hours, are the most important in locating the colonic pathology, for by that time we are able to visualize relative changes in the contour of the large bowel, and the final progress of the barium into the rectal pouch. By such examinations we are absolutely able to locate the position of the band, the degree of ptosis of the transverse colon, its relative position to the ascending colon (Figs. 10 and 11), and the degree and location of the bowel obstruction. With such findings our diagnosis is complete.

The treatment, of course, is the surgical relief of a mechanical condition. An incision is made that will best expose the entire pathology, and depends upon its extent. By careful dissection the adhesive bands are freed. It is rarely necessary to resect any portion of the tissue, because there is an immediate contraction as soon as it is liberated. Due to the fact that there is such a close agglutination between the adhesive bands and the peritoneal covering of the gut, it is impossible to separate the two without injuring the peritoneal coat. All such raw surfaces must be peritonized, otherwise a reformation of adhesions will occur, practically the same mechanical condition will be present, and the patient will experience little, if any, relief.

The postoperative treatment of these cases also is of prime importance in preventing the reformation of adhesions. For several days following operation, they are kept on their left side as constantly as is possible. This has the mechanical effect of allowing the newly freed transverse colon to drop away from its former position beside the ascending portion. After time enough has elapsed for the bowel to become again peritonized, this is not necessary. As a further effort to prevent new adhesions, active movement of the bowel is kept up by the frequent administration of cathartics, preferably magnesium sulphate. This has the disadvantage of increasing gas accumulation within the bowel, but the slight bowel distention which results from the gas is an added mechanical advantage against any new adhesions. However, the patients must be watched closely, for too great a distention is a forerunner of a paralytic ileus.

SUMMARY

1. The condition seems to be a definite surgical entity.

2. Its symptoms simulate those of chronic appendicitis, but often occur after appendectomy.

3. Chronic appendicitis is sometimes an accompanying condition, but is not the causative factor.

4. Evidently it is an inflammatory lesion due to colonic stasis.

5. An absolute diagnosis may be made by means of roentgenographic examinations, and in no other manner.

6. The treatment is the surgical relief of a definite mechanical condition.

PLACENTA ACCRETA; ITS INCIDENCE, PATHOLOGY, AND MANAGEMENT[1]

By JOHN OSBORN POLAK, M.D., F.A.C.S., and GEORGE W. PHELAN, M.D., Brooklyn

At the meeting at White Sulphur Springs, I presented to this society a preliminary report on the management of the third stage of labor, which was based upon a study of 2,000 deliveries. In this paper I made some suggestions as to the management of the retained and adherent placenta and recommended that in placenta accreta, hysterotomy followed by hysterectomy should be the procedure of choice. The suggestion, however, must have fallen upon deaf ears, for it was not even mentioned in the discussion.

Recent studies of the mortality incident to true adherent placenta, both here and abroad, by obstetric methods heretofore in vogue show that placenta accreta is attended by a high death toll. True placenta accreta is extremely rare, but four such cases have been encountered by the writer in 30 years of obstetric practice; yet during 20 years it has been my fortune to be at the head of three active services. These cases present a definite anatomical and clinical picture, which, if it were thoroughly understood, would considerably reduce the mortality.

It is the purpose of this short contribution to show first, that, though rare, placenta accreta is a pathological entity and should not be confounded with adhesion of the placenta, for an accreta is the result of an entire or almost entire absence of the decidua basalis which exposes the muscle of the uterine wall to the erosive action of the trophoblast and penetration of the villi. This intimate union of the placenta and muscle wall makes it impossible to find any line of cleavage for placental separation.

Second, that the etiology is dependent upon changes which produce an atrophy or absence of the normal uterine decidua, such as previous manual removal of the placenta, vigorous curettage, endometritis, submucous myomata, etc.

Third, that the high mortality attending this complication is the direct result of improper treatment from failure to recognize that in a true accreta, there is no line of cleavage, for the placenta is not only an intimate part of the muscular wall, but the erosive action has so thinned this wall that attempts at removal produce hæmorrhages and open up avenues for infection and even permit perforation of the uterus.

Finally, that true placenta accreta necessitates both rational and radical management with attention to every aseptic detail.

In order to establish the truth of these statements, we have reviewed 6,000 deliveries occurring in the in- and out-patient services of the Long Island College Hospital. These have been studied to determine (a) the frequency with which the complication is met; (b) the incidence of infection; (c) the method of treatment employed and, finally, the complications that have occurred including the mortality. The existence of abnormal adhesion has been assumed when the placenta has been retained within the uterus for more than two hours following the delivery of the child, without the occurrence of uterine hæmorrhage, for, clinically, separation of the pla-

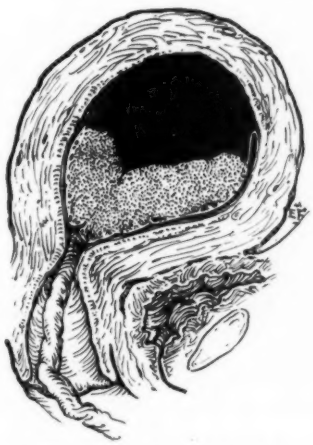

Fig. 1. Retained placenta.

[1] Read before the American Gynecological Society, Hot Springs, Virginia, May, 1923.

Fig. 2. Case of adherent placenta. Cornual implantation.

centa does not occur without bleeding. It has been necessary to differentiate this condition from simple retention of a separated placenta, due to the partial closure of the retraction ring.

In retention of the separated placenta, three clinical signs are always evident: (1) uterine bleedings; (2) descent of the cord; (3) the characteristic ball-like condition of the fundus; rise of the fundus is not found in either retention or adhesion.

Conversely, it may be stated that in adherent placenta or placenta accreta, provided there has been no manipulation to cause partial detachment, there is neither hæmorrhage, descent of the cord, or change in the position of the fundus, and the fundus assumes a characteristic shape, being broader

Fig. 3. Formation of placenta.

from side to side and intermittently relaxed, not assuming the firm contraction and ball-like shape characteristically present in the separated placenta.

If we revert for a moment to the consideration of the formation of the placenta, we will remember that it is formed partly from the chorion frondosum and the decidua basalis, and that the villus processes of the chorion are in contact with the basal decidua, grow rapidly, and penetrate by erosion into this decidua, while those on the remaining portions of the ovum, in contact with the uterine decidua, atrophy.

Histologically the chorion frondosum consists of a connective-tissue layer which lies next to the amnion and a villous layer made up of villi which are covered by an outer layer of trophoblastic cells; as each villus grows into the basal decidua which protects the muscular structure of the uterus from invasion, it makes for itself a space by erosion into the mucosa. This space is always larger than the villus which is growing into it and ultimately forms a spacious sinus or blood space which is filled with maternal blood. Into this sinus the villus projects and becomes bathed in the maternal blood, but does not penetrate beyond the protective basal decidua. These are called the floating villi; others, however, opposite the funic insertion and near the

Fig. 4. Engorged blood vessels in serotina.

Fig. 5. High power of same field.

placental circumference, become more deeply attached and extend further into the mucosa, and fasten the placenta to the underlying structures. These are the anchoring villi. The protection of the muscular wall of the uterus from the diffuse erosion of these villi, covered as they are with trophoblastic cells, is due to the fact that normally there is interposed a protective layer of decidual reaction in the basalis or serotina. Certain conditions cause this protective layer of endometrium to be absent or atrophied. This permits the villi to erode themselves into the muscular wall of the uterus and even penetrate through the uterine muscle. This fetal cell invasion so weakens the uterine wall that perforation is easy; the placenta and myometrium become a continuous mass inseparable from one another. This absence of the decidua basalis and implantation of the placenta in the myometrium must not be confused with prolonged adhesion of the placenta due to muscular difficulties, as when implantation of the ovum occurs in the tubal corner where the decidua is often absent or poorly developed; or when implantation has taken place on a uterine septum; or when the placenta is large and thin as in certain instances of twins or hydramnios with a fundal attachment. In these cases the placenta does not become separated in the usual way, yet we find that the histological structures of the basalis can be demonstrated.

If we go a step further and review the mechanism of placental separation, we can readily see how dependent we are upon the serotina for accomplishing this separation. Immediately on the delivery of the fetus, the entire uterus, except the placental site, retracts and thickens. This immediately produces an enlargement and engorgement of the vessels in the site, within the spongy layer of the serotina. With the first contraction, the placental area is suddenly reduced and more vessels are torn by the folding or puckering of the placenta, and retroplacental bleeding takes place, which with the next contraction is compressed along the line of cleavage and the placenta separates off in the spongy layer of the serotina. With the absence of the serotina with its spongy layer, which cannot be found in histological study of placenta accreta, the villi penetrate the muscle walls of the uterus and hence the placenta and walls make up one continuous structure, and present no line of cleavage.

In reviewing our records of both in- and outpatient services for the past 5 years, we find that there have been eight manual removals of the placenta; three were partially or completely adherent, but a line of cleavage could be found and followed until separation was completed; four were separated but retained

Fig. 6. Showing separation of placenta by retroplacental blood accumulation.

by a retraction ring, while in one no line of cleavage could be found or demonstrated. Placental tissue was removed piecemeal; the removal was incomplete and the hæmorrhage was so excessive that further manipulation had to be postponed, the uterus firmly packed, and restoratives applied. A donor was sought, but before transfusion could be done, patient died, 9 hours after admission. Autopsy was denied. It is fair, therefore, to place the incidence of accreta at approximately one in six thousand (1:6000).

In my private series, the incidence has been greater; but considering that only cases with complicated histories and in difficulties reach the obstetric consultant, the fact that three placentæ accretæ have been met should not be considered exceptional. In two, manual removal was unusually difficult and this piecemeal removal was attended with such severe hæmorrhage as to cause me to desist and pack the uterus. Both died of sepsis. Both of these women were multiparæ and had had previous manual removal which was also a fact in the fatal case reported by Dietrich, and the successful one of Greifswald.

From a study of histories of reported cases available, it is apparent that previous manual removal of an adherent placenta or repeated or vigorous curettage predisposed to adhesion in subsequent pregnancies.

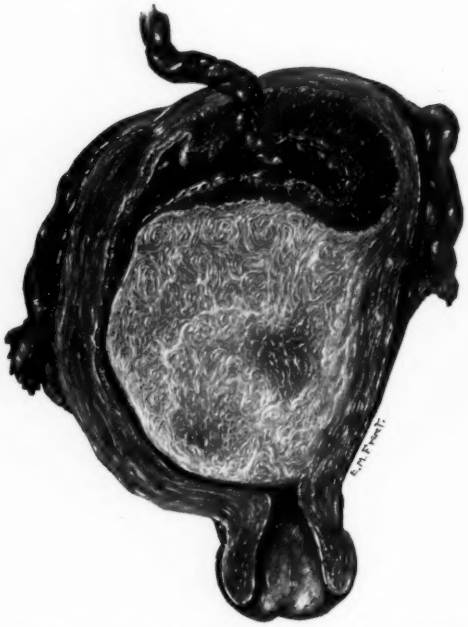

Fig. 7. Case of placenta accreta. Implanted in uterine cornu, and on atrophic endometrium, over large myoma.

My third case is illustrative of an accreta occurring on an absent decidua, a result of repeated curettage with an implantation on the atrophic endometrium of a submucous myoma.

This patient was 26 years old and married 5 years; never pregnant. She had been subjected to four curettings, one for sterility and three for menorrhagia. There was no menstruation after her last curetting. When seen by the writer 13 months later she was in labor; the uterine tumor was very large, reaching the ensiform, and a smooth hard tumor mass could be palpated just above the pubis in the left lower quadrant; the labor was easy and a dead born, 7-months fetus was delivered after about 5 hours of pain. Following the birth of the child the fundus remained about 10 centimeters above the umbilicus. The placenta was not expelled. There was no bleeding. The uterus contracted intermittently. Repeated efforts with Credé's method failed to express the placenta. She was removed to the hospital and 6 hours after delivery under ether narcosis and with the strictest asepsis the gloved hand was introduced into the uterus. In the lower segment on the left uterine wall just above the internal os was a large submucous fibroid (the size of a grapefruit) spread out on its summit and attached to the entire left uterine wall. Above the tumor was

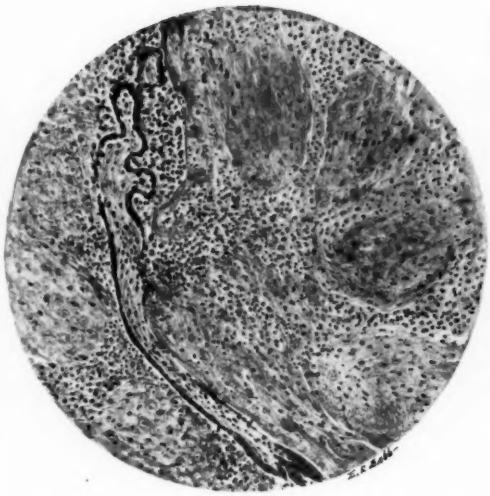

Fig. 8. Placenta accreta. Villus penetrating muscle.

spread the placenta. No line of cleavage could be detected; so we decided that as it was a clean case we would open the abdomen, do a hysterotomy, attempt detachment under sight, and, if we failed, remove the uterus. This was done after incising the uterus to the right edge of the placenta; an attempt at separation was made, but no line of cleavage could be detected. The uterine wall over the placenta was relaxed and thin; a supracervical hysterectomy was made and an uneventful recovery followed.

Gross study of the uterine wall showed that the uterine muscle had been replaced by placental tissue, and at several points it had only a peritoneal covering. This was most apparent just opposite the insertion of the cord. Microscopically there was an absence of the decidua, the villi were directly attached to the uterine muscle and in several fields syncytial cells were found in the fibers, while masses of the same cells had split the muscle wall into fragments. Study of this specimen shows the futility of attempting manual removal in placenta accreta and emphasizes the importance of aseptic exploration before proceeding to traumatize these friable tissues.

Placenta accreta, therefore, may be described as the implantation of the ovum and the development of the placenta on a uterine wall in which there is a total or almost total absence of the decidua serotina. The chorionic

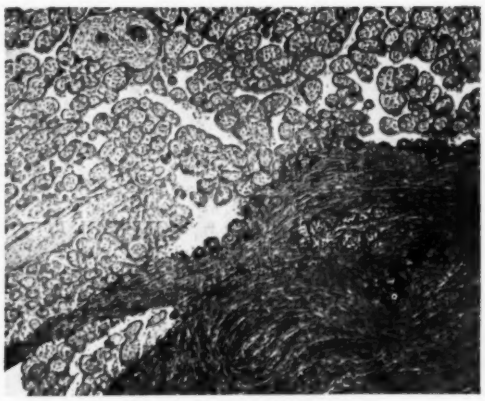

Fig. 9. Placenta accreta invasion of syncytial cells into the muscle wall.

villi erode themselves into the muscle fibers, hence the uterine muscle partly degenerates resulting in a thinning of the uterine wall over the placental site, even to the point of rupture.

An appreciation of this pathology shows why the mortality from hæmorrhage, sepsis, and perforation, make this condition so formidable. Of our four cases, three died from hæmorrhage or sepsis; one recovered. This with Greifswald's case are, we believe, the only two recoveries in the literature.

CONCLUSIONS

1. In our series the incidence of placenta accreta is about 1 in 6000 cases.
2. There is considerable confusion in the minds of the profession between simple adhesion of the placenta and true accreta.
3. Accreta is a definite pathological entity.
4. Manual removal is impossible and can only result in hæmorrhage, sepsis, or perforation.
5. Every delayed placenta with no hæmorrhage should be viewed with suspicion; and no attempts at Credé's method should be made if the clinical signs of separation are not present.
6. In the presence of an attached placenta without bleeding, aseptic exploration under anæsthesia should be made to determine the subsequent procedure.
7. Finally, if no line of cleavage can be demonstrated, hysterectomy should be done.

PAPILLARY TUMORS OF THE RENAL PELVIS

By ALBERT J. SCHOLL, M.D., Rochester, Minnesota
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PRIOR to the advent of modern cystoscopic methods, the study of renal tumors was confined to necropsy findings; their clinical recognition was unusual. Gurlt, in 1880, reviewing 14,630 cases of malignancy from Vienna hospitals, found only 16 clinically recognized renal tumors (1 in 914), while Reiche, in 11,930 necropsies in cancer patients, found 80 (1 in 149). Albarran and Imbert, in a review of the literature up to 1903, were able to collect only 585 cases of renal tumor. Taddei collected 435 cases published from 1902 to 1907. Tumors of the renal pelvis were even more difficult to recognize clinically than the large, bulky, parenchymatous growths; the majority of the cases had been discovered at necropsy. Forty-two of Albarran's cases of renal tumor, 31 of Taddei's, and 9 of 652 cases collected by Kuster were primarily of the renal pelvis. Since then, collected reviews of a comparatively large number of cases of tumor of the renal pelvis have been reported by Stuesser, Mock, and Lower. Other reports have been limited to special pathological and clinical types; Savory and Nash, Judd, McCown, and Hryntschak reviewed cases of papillary growth. Kretschmer, in 1917, gave an excellent review of non-papillary tumors. Spiess, in 1915, was able to collect 136 cases of various histological types. From 1905 to 1922 there were 273 cases of renal tumor treated at the Mayo Clinic, 13 of which were primarily of the renal pelvis, 8 being papillary epithelioma.

The majority of tumors of the renal pelvis are of papillary origin. In 18 of Albarran's 42 collected cases, in 21 of Taddei's 31 cases, and in 76 of Spiess' 136, the tumors were of this type. Recently a number of cases of papillary tumors removed surgically have been added to the literature. Hryntschak, in 1920, collected 68 cases of definite papillary growth, many of them surgical.

ETIOLOGY

Ewing describes the disease colitis polyposa as an example of the transition from an inflammatory overgrowth to a malignant tumor. In 50 cases collected by Doering, there were 37 deaths; malignant changes were found in 31. Similar changes occur in the urinary tract; long-standing irritation and inflammation are very probably factors in the formation of papillomata and epitheliomata. Rehn, in 1895, and Seyberth, in 1907, reported cases of papillary tumors in the bladders of dye workers, a result of chemical irritation. Goebel reported similar cases, the result of mechanical irritation of encysted bilharzia organisms. In the renal pelvis various irritative conditions, such as infection and urinary concrements probably result in similar inflammatory overgrowths. In one case observed in the Mayo Clinic, nephrectomy was performed for severe renal infection. Marked inflammation of the pelvic mucosa and a definite villous pyelitis were found (Fig. 1). There was also an area of leucoplakia of the pelvic mucosa (Fig. 2). Both of these conditions suggest inflammatory irritation. In another case, villous inflammation and stone were found. In both of these cases there was marked round-cell infiltration, œdema, and fibrosis of the surrounding tissue. Calculi apparently are not important in the etiology of papillary tumors. Only a few cases of stone with papilloma have been reported; the stone is apparently a result of the same irritative factor which causes the epithelial proliferation. Kohlhardt also described a case of villous pyelitis, the result of long-standing inflammation. Orth believes that this villous or papillary inflammation may merge into, and cannot be histologically distinguished from papillary tumors. In the examination of the grossly uninvolved mucosa and submucosa adjacent to papillary tumors of the renal pelvis, small, villous, apparently inflammatory processes are often found differing from similar villous processes that are associated with infection or stone, which are confined to the epithelial layer. The papillary processes associated with tumors sometimes involve the submucous tissue (Fig. 3). Kauf-

Fig. 1.

Fig. 2.

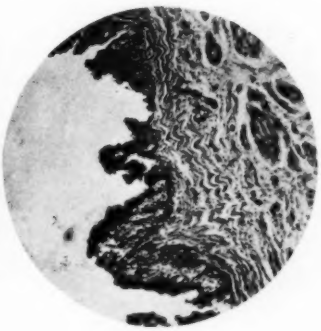

Fig. 3.

Fig. 1. Villous papillitis. Histologically similar to conditions found in association with papillary tumors of renal pelvis. (A126632.) (× 100.)

Fig. 2. Leucoplakia of the renal pelvis which was associated with villous papillitis. Both conditions probably resulted from the same irritative factor. (A126632.) (× 50.)

Fig. 3. Early stage in formation of papilloma of renal pelvis. Section from mucosa adjacent to papillomatous masses. (A287879.) (× 50.)

man attributes the development of papilloma to chronic exudative inflammation. He cites the case of a patient with a fistula between the renal pelvis and the duodenum caused by the sloughing of a renal stone into the intestine. For years particles of undigested food were passed through the urethra. At necropsy the ureter and renal pelvis were found to be greatly thickened, inflamed, and covered with small papillomata.

In most areas the mucosa between the papillary growths shows evidence of inflammation on the surface, but in some cases it is apparently normal. The submucous tissues almost invariably show the effects of inflammation: round cells and plasma cells, granulation and fibrous tissue, with many large thin-walled blood vessels. Stoerk believes that the free epithelial surface, injured by long-continued, toxic, or mechanical irritation which results in a regenerating fibrosis, is an etiological factor in the development of papillomata. The large blood vessels grow up toward the surface, branch out, and are covered with epithelium. Undue proliferation of epithelium and regenerating vessels eventually result in the tufted villous masses.

PATHOLOGY

In the early stages the tumors are small, often multiple, flat or thickly pedunculated and confined to the renal pelvis (Fig. 4). They spread rapidly and extensively, involving the calyces and sometimes the ureteral outlet. As a result of obstruction or invasion, the renal cortex may become extensively atrophied. In the late stages the kidney becomes a distended, sacculated, often infected mass with complete loss of function. In cases reported by Poll and Dickinson, the entire pelvis and calyces were covered with fine villous masses. The kidney is generally moderately enlarged with slight pelvic dilatation, or the mass may be converted into a large dilated sac. The papillomatous growth may be felt through the pelvis as a soft doughy mass. It is sometimes impossible to detect anything abnormal from the external appearance, as in a case described by Bruett. Derewenko, in a similar case, opened the kidney and explored it digitally, but found no growth. Seven months later it was necessary to remove the kidney on account of severe bleeding; four papillomata were found in the renal calyces.

Since the renal pelvis does not offer a free space for growth like that of the urinary bladder, the pelvis is rapidly filled and the papillomatous masses become matted together under tension so that they bulge from the pelvis when the kidney is opened. The ureter may be involved by direct extension, or by transplants which may stop at any of the normal constrictions (Fig. 5). The most common site is the ureterovesical juncture; here the growth

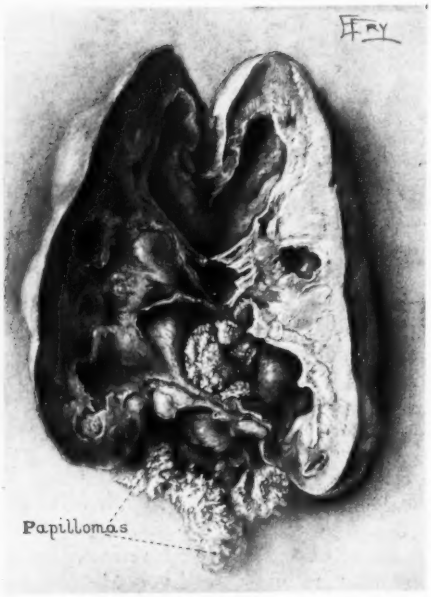

Fig. 4. Small pedunculated papillomata of the pelvic mucosa.

may project into the bladder and invade the surrounding mucosa. Occasionally the growth completely encircles and occludes the ureteral lumen. Often the ureter is dilated and thickened, and the uninvolved mucosa shows evidence of chronic inflammation. As in the mucosa of the renal pelvis, there is an extensive round cell infiltration, œdema and moderate fibrosis of the submucous tissues. Extension to the ureter was found in 6 of Albarran's 18 collected cases.

In both the renal pelvis and the ureter the individual papillomatous fronds are shorter and broader than similar growths in the bladder; there is a more extensive fusion of adjacent fronds, and atypical cell masses are more often seen (Figs. 6, 7, and 8). Histologically the growth in the ureter and pelvis contains more areas undergoing malignant degeneration than the comparatively more villous transplants in the bladder, and loss of cellular polarity and regularity, with the presence of numerous mitotic figures, may occur in a grossly benign tumor, as in papillomata of the bladder. In certain cases the histological difference in the tumors in the bladder, ureter and renal pelvis is only slight. Lower reported a case in which sections from the kidney, ureter, and bladder were histologically similar.

The majority of papillary tumors of the renal pelvis are histologically similar to the malignant papillomata, or the papillary epitheliomata occurring in the urinary bladder. In an occasional case glandular changes have been noted in cells of the papillary structure. Grohé reported a case of adenocarcinoma of the renal pelvis; the pelvis also contained a number of small papillary excrescences. Malignant papillary adenomata of the body of the kidney of the type reported by Judd may penetrate to the pelvis, in which case the site of origin is questionable. Hryntschak considered only 6 of his 68 collected cases as definitely benign, basing his opinion in most instances on clinical data. Morris asserts that even though villous growths in the renal pelvis may be histologically benign, they are far from being clinically benign. If a careful histological study is made of sections from various parts of the growth, small circumscribed areas of malignancy will almost invariably be found, even in the small, pedunculated, grossly benign papillomata. In spite of the histological report it is impossible definitely to determine, after an examination of the initial lesion, what the outcome will be. Bruett, who observed several cases for a number of years, says that papillomata which, after prolonged observation, prove themselves unquestionably benign, are very rare. A number of reported cases, apparantly benign primary lesions, have been followed by malignant extensions or recurrences shortly after operation. Barth reported a benign papilloma of the pelvis with metastatic cancer in the regional glands. Reynes removed a kidney for a benign growth which recurred as cancer 2 years later. In a case of Israel's there was a benign growth in the renal pelvis and bladder, which later recurred as cancer in the scar of operation. Nephrectomies for benign lesions with development of generalized carcinomatosis are reported by both Derewenko and Pantaloni. Late recurrences also occur. Tikhoff reported a case of recurrence in the scar 10 years after the removal of a bening

papilloma, and Bland-Sutton, a similar case, 11 years after the original operation. In contrast to this, recurrences occasionally develop very rapidly and extensively. Asch removed a kidney with a small papilloma of the pelvis. Two months later there was a secondary deposit in the bladder about 7 centimeters in diameter. The bladder was resected: 6 weeks later the patient died and metastasis to the scar was found. Zuckerhandl, in a similar case, noted the recurrence of twenty tumors, 1 or 2 centimeters in diameter, in the bladder 6 months after the removal of a benign papilloma of the kidney. The bladder was not involved in either case at the first examination. On the other hand, Drew and Barker report malignant lesions in the kidney with benign secondary deposits. Possibly the primary exciting cause continued, resulting in further hyperplasia of the original focus after the occurrence of extension to the bladder.

Fig. 5. Section of ureter and bladder wall containing transplants from papillary carcinoma of renal pelvis.

DIAGNOSIS

Hæmaturia is the most common and often the only symptom observed in the early stages of tumor of the renal pelvis; it is generally profuse, continuous, or intermittent, with only short intervening periods, and lasts from several days to several weeks. In papillary tumors the excessive vascularity and vulnerability result in severe hæmorrhages which often follow slight injury or violent muscular movements. The bleeding may almost exsanguinate the patient, as in a case described by Blum. Morris reported a patient with a papilloma of the renal pelvis who died from severe hæmorrhage of 7 months duration. The hæmorrhage occurring with tumors of the renal parenchyma is generally of short duration and irregular, with free periods of several months. The bleeding called "essential hæmaturia" may be of long duration, but it is generally not consistently profuse, and the passing of blood clots, common with papillary tumors, is comparatively rare. In tumors of the pelvis the function in most cases is markedly reduced; in essential hæmaturia it is generally normal. Braasch says: "If cystoscopy reveals a papillary tumor of the bladder, together with a unilateral renal hæmaturia, the probable diagnosis would be pelvic epithelioma with vesical metastasis. When there is no tumor of the bladder the diagnosis becomes more difficult. In the majority of cases the diagnosis can be made quite certain by means of pyelography. In the first place, if the growth is large enough, it may occlude the pelvis entirely. Usually, however, there is an irregular, partially obliterated outline of the pelvis together with dilatation or elongation of one or more calyces. It must be remembered that partially organized blood clots in the renal pelvis may cause an outline simulating filling defects." Thomas, Colston, and Landon and Alter report cases of papillary tumors in which such a defect was noted.

Pain is not a prominent symptom, and when present it is generally dull and constant. There may be attacks of sharp renal colic associated with the passage of blood clots, or

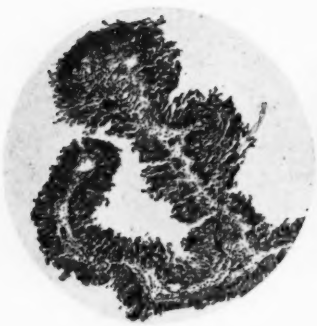

Fig. 6.

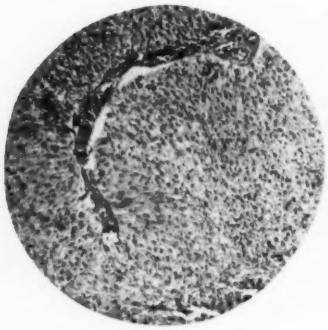

Fig. 7.

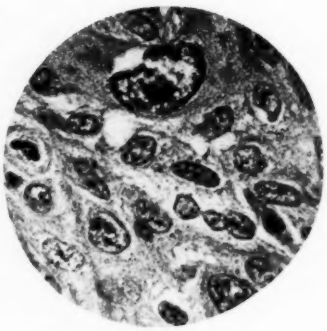

Fig. 8.

Fig. 6. Delicate arborescent papilloma of bladder; transplant from papillary epithelioma of renal pelvis. (A212016.) (× 50.)

Fig. 7. Fusion and matting together of adjacent fronds in papillary tumor of the renal pelvis. (A287879.) (× 120.)

Fig. 8. Atypical cell forms in rapidly growing papillary carcinoma of the renal pelvis. (× 500.)

due to a transient hydronephrosis, or hæmatonephrosis resulting from ureteral obstruction. The obstruction may be caused by the passage of blood clots or fragments of tumor, or by ureteral kinking from ptosis of the heavy kidney. Israel calls attention to the occasional presence of an intermittent abdominal tumor, a hæmatonephrosis due to ureteral obstruction and bleeding. The passage of a blood clot or papillomatous fragment may be followed by large quantities of bloody urine, coincident with the disappearance of the tumor and relief from pain. Lion reported a case of long-standing renal colic which ceased following the passage of a fragment of tumor and great quantities of urine. Stoerk, and Hoché and Damage report the passage of multiple fragments of tumor in the urine. The pelvic dilatation resulting from the obstruction is sometimes very great. Cope reports the case of a pelvis holding 2 pints and Reynolds of a renal distention of 14 pints, both associated with tumors of the renal pelvis.

The duration of symptoms, in most cases of papillary tumors, is comparatively short, generally from 6 to 12 months. Occasionally the symptoms, especially the attacks of hæmaturia, are of many years' duration. Busse reported the case of a patient who had hæmaturia for 20 years. Thornton reported a case of attacks of renal colic for more than 21 years, and intermittent hæmaturia for 9 years.

Cystoscopic examination may reveal a normal bladder. Sometimes a small papillomatous tuft is found protruding from the ureteral orifice, or there may be a number of papillomatous masses, or a single, flat, rounded growth surrounding or completely occluding the ureteral opening. In certain cases numerous small growths are found extensively scattered over the mucosa of the bladder, or there may be one or more minute papillomata directly in line with the ureteral outflow. The secondary tumors in the bladder are usually flat and low, and do not have the wavy arborescence and villosity which occurs with primary papilloma of the bladder of the same size. In some cases extensive secondary transplants in the bladder may completely cloud the primary renal or ureteral origin, especially if the ureteral orifice is obscured by the tumors. Occasionally, in resection of the bladder, presumably for a primary tumor, the lower portion of the ureter is cut across and papillomatous masses are found protruding from the cut ends, giving a clue to the renal involvement. This may sometimes be misleading, as the growth may originate primarily in the bladder and extend upward through the ureteral opening. In a case observed at the Mayo Clinic an extensive papillary epithelioma in the region of the ureter was removed, with the lower 3 centimeters of the ureter which was moderately dilated. A few tufted papillæ were found protruding from the cut

end of the vesical portion of the ureter. The upper end was ligated and dropped back in the wound; later the remaining portion of the ureter and the kidney were removed. The kidney was markedly atrophied from infection and distention, but there was no evidence of tumor, either in the kidney or remaining ureter.

PROGNOSIS

Due to the greater friability and more frequent fragmentation, the villous forms of papilloma develop transplants more readily than the flat or more malignant types. Of 25 cases collected by Mock, in which operation was performed, and the growth considered papillary carcinoma, 23 recovered from the operation. Eighteen of the patients were traced after operation; of these, only 5 remained well; the others died or developed recurrences.

In papillomatous tumors, recurrence is almost a certainty, unless a complete ureterectomy, including resection of the ureteral orifice is carried on, either at the time the kidney is removed, or later. The transplants to the bladder may be exceedingly numerous and persistent, necessitating frequent, regular, cystoscopic examinations with prompt treatment of all newly developing growths. The 8 cases of papillary tumor of the renal pelvis reported were treated at the Mayo Clinic between February, 1910, and February, 1922, the early history of 3 being reported by Judd in 1919.

REPORT OF CASES

CASE 1. (A383357.) H. M. H., a man, age 48, came to the Clinic February 6, 1922. One year before, following trauma, he had had sudden pain in the back; 2 days later blood was passed with urine. This bleeding continued with only a few intermissions up to the time of his registration at the Clinic. When he remained quiet the urine was but slightly stained, but when he was moving about freely the urine resembled pure blood. There was no pain, colic, dysuria, or frequency. He had not passed stones or blood clots. He felt that he was gradually becoming weaker. He had been cystoscoped and a small papilloma of the bladder had been fulgurated 10 months before, and 6 months later a recurrence in the bladder was also fulgurated, and the pelvis of the right kidney lavaged with silver nitrate on account of bleeding from the right side.

Physical examination was negative. The urine contained a large amount of blood, but no pus. The

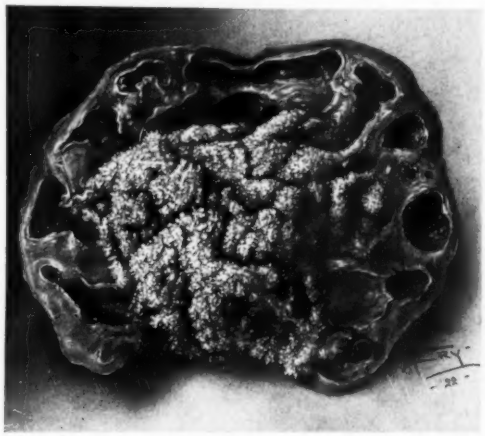

Fig. 9. Extensive papillary carcinoma of the renal pelvis in a completely destroyed kidney. The ureteral outlet obstructed; no transplants in ureter or bladder. (A383-357).

hæmoglobin was 50 per cent, and the erythrocytes 3,500,000. The phenolsulphonephthalein test was 60 per cent, and the blood urea 34 milligrams for each 100 cubic centimeters of blood. Cystoscopic examination revealed hæmorrhagic urine. There was a villous papilloma 2 by 2 centimeters on the left posterior wall of the bladder. The left ureteral orifice was normal. There was periodic dribbling of pure blood from the right orifice. Urine was not secreted from the right side in 5 minutes.

At operation, February 16, right nephrectomy and partial ureterectomy was performed. The renal pelvis was dilated and found to contain a soft mass; very little renal tissue remained. The ureter was cut with the cautery, ligated, and dropped back. On account of evidence of infection the remainder of the ureter was not removed. Following operation, the patient developed thrombophlebitis of the right leg and remained in the hospital 44 days. Further treatment of the bladder was delayed several months. Cystoscopic examination June 1, revealed two small papillomata of the bladder; these were completely fulgurated twice. The right orifice was not seen.

June 26, through a right rectus extraperitoneal incision, the remaining portion of the ureter and a segment of the wall of the bladder were removed with the cautery. The ureter was very adherent. The patient convalesced uneventfully and left the hospital on the fourteenth day.

The kidney was large, distended and thin walled. The entire pelvis was filled with a large, bulging, papillomatous mass (Fig. 9). In areas not covered by the growth, the mucous membrane of the pelvis and calyces was markedly thickened and fibrosed. The kidney weighed 275 grams. The ureter was completely blocked by the tumor at the ureteropel-

Fig. 10.

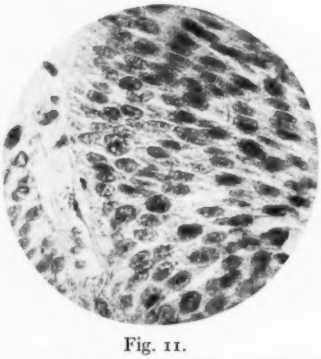

Fig. 11.

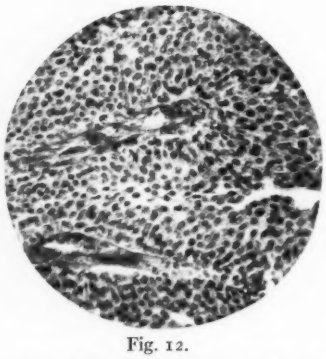

Fig. 12.

Fig. 10. Solid papillary carcinoma of the renal pelvis. (A383327.) (× 50.)

Fig. 11. Area of localized malignancy in a papilloma of the renal pelvis. (A383327.) (× 350.)

Fig. 12. Fusion and matting together of adjacent fronds in papillary tumor of the renal pelvis. (A349145.) (× 120.)

vic juncture. The lower segment of the ureter and ureteral orifice were not abnormal. The growth was a malignant papilloma composed of branching, papillomatous masses. There was extensive fusion of adjacent fronds and a number of areas with definite, malignant changes. These areas were made up of large, irregular, round cells, with many atypical mitotic figures and a lack of the usual elongated cellular regularity and disposition (Figs. 10 and 11).

CASE 2. (A349154.) J. M., a man, age 61, came to the Clinic February 18, 1921, on account of pain in the left kidney area of 2 years' duration. The pain was dull and heavy, and came in attacks lasting for weeks. He frequently passed coffee-colored urine, and had lost considerable weight and strength. The pain had been almost constant for the past 6 weeks, and for the past 3 weeks a mass had been noted in the left abdomen.

Examination revealed a large, irregular, nontender, cystic and freely movable mass extending about 6 centimeters below the left costal margin. Ballottement could be obtained between the costovertebral angle and the anterior abdominal wall. A moderate sized varicocele was found on the left side. The urine contained pus and blood, and the renal function was 60 per cent. Roentgenographic examination of the stomach, colon, and urinary tract was negative. Cystoscopic examination revealed a few minute papillomatous tags projecting from the left meatus. The right ureteral opening was normal, and normal urine was obtained on catheterization. The left opening was contracted, but normal appearing urine was being secreted. On catheterization of the left ureter, hæmorrhagic urine was obtained, and there was no phenolsulphonephthalein return from this side in 15 minutes. The right kidney drained poorly, but the phenolsulphonephthalein return was fair.

At operation, February 23, the kidney was found to be hydronephrotic and soft, and the renal tissue mostly destroyed. The ureter which was apparently normal, was cut, ligated, and the lower segment dropped back in the wound. There was evidence of a tumor near the spine, but the patient's condition contra-indicated further exploration. The patient had no trouble following operation, and left the hospital on the twenty-third day. Two weeks later he was given a complete roentgen-ray radiation of the involved area. He felt better immediately after the operation, but rapidly lost strength and weight and died 6 months later.

On section, the renal pelvis and calyces were found to be almost completely covered with stubby papillomatous masses. The growth was a papillary epithelioma, composed of flat, irregular masses of epithelial cells. In a few areas the papillary outline and the axis of the central connective tissue was retained (Fig. 12). The cells were large, with clear outlines and distinct nucleoli; they were arranged without any definite formation or polarity. There was a tremendous thickening and fibrosis of the pelvic wall, with extensive plasma cell and round cell infiltration; there were also numerous scattered areas of tumor cells which filled many of the underlying blood vessels and lymph spaces. In the few areas not involved in the process, the pelvic mucosa was thickened and irregular, merging gradually into adjacent masses.

CASE 3. (A338759.) R. M., a man, age 46, came to the Clinic October 23, 1920, because of attacks of painless hæmaturia that had existed the past 4 years. The attacks lasted 2 or 3 days and recurred every 2 or 3 months. The attacks were longer and more frequent when the patient was working on his farm. While resting the hæmaturia almost disappeared. During the last 2 years urination had been moderately frequent. For the last 2 months a dull heavy pain in the right lumbar region had kept the patient from working. There had been no loss of weight, and his appetite was normal.

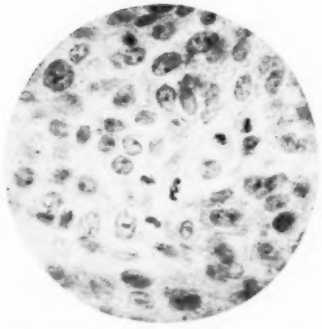

Fig. 13. Rapidly growing transplant to bladder from papillary epithelioma of renal pelvis. Numerous mitotic figures are present. (A338759.) (× 450.)

Fig. 14. Papillary carcinoma of the renal pelvis. The ureteral outlet was patent, permitting numerous transplants to pass to the lower ureter and bladder. (A256716.)

The physical examination was negative, but the urine contained large amounts of blood, and the hæmoglobin was 75 per cent. Roentgenograms of the kidneys, ureters and bladder revealed nothing abnormal, and the phenolsulphonephthalein test was 45 per cent. At cystoscopic examination multiple flat papillomata of the bladder, involving several distinct areas, especially the left orifice were found.

November 23, the bladder was opened suprapubically and a large, flat papillary growth removed from the left wall and base, together with a number of smaller tumors. One small growth was also excised from the right base. The left ureter opened in the center of the large growth. In cutting across the ureter, 3 centimeters above the bladder, small papillomatous masses protruded from the cut end, indicating a possible involvement of the upper urinary tract. The patient had an uneventful convalescence, and on the fourteenth day (December 6, 1922) a left nephrectomy and ureterectomy was performed. The kidney was found to be moderately hydronephrotic and the pelvis was distended with a soft tumor. The patient left the hospital 17 days after the second operation with the kidney wound healed. He remained under observation for 6 weeks, during which time both wounds healed completely. Several months after leaving the Clinic, his general health began to fail, and he died 9 months after the first operation, presumably from recurrence.

The kidney weighed 270 grams. It was small and fibrosed, with a moderate semi-solid pelvic dilatation. On section, the tissue was found to be markedly thinned; the calyces were extensively dilated and filled with an extensive, flat papillary growth. The pelvic wall was greatly thickened, but in most areas it was macroscopically free from tumor. The ureter was greatly thickened and dilated, and sharply kinked in several places. It was free from tumor save at the ureterovesical juncture where an area 3 centimeters in length, almost completely blocking the lumen, had been cut through at the first operation. In the sections of the ureter removed at the first and second operations, the growth was flat and composed of short, stubby, papillomatous masses with very little branching or free arborization. The largest tumor removed from the bladder measured 7 by 5 by 2 centimeters and was composed of firm, compact, flat, papillary masses of the type usually described as papillary epithelioma. Histologically, the sections from the pelvis, ureter, and bladder confirmed the diagnosis of solid papillary epithelioma. The cells, which were large and irregular in size and took the stain deeply, were massed together with only a small amount of supporting tissue. Remnants of altered papillæ indicated the original papillary structure. In the kidney the tumor had broken into and extensively involved the remaining parenchymal tissue. The malignant cells from the growth in the bladder were larger and more irregular than those from the kidney or ureter; the process had evidently been more rapid in the bladder (Fig. 13). There was widespread round-cell infiltration, with a moderate fibrosis in the bladder wall, but no evidence of malignant extension through the bladder wall was found.

Case 4. (A67588.) W. S. D., a man, age 65, came to the Clinic April 9, 1919, complaining of hæmaturia of 2 years' duration. It occurred in spells lasting several days, once every 4 or 5 months. There had been sharp, severe pain in the region of the left kidney; recently this had developed into a constant ache. Six months before, a small stone had passed through the urethra without pain.

The physical examination was negative except for tenderness in the upper left abdomen. The urine

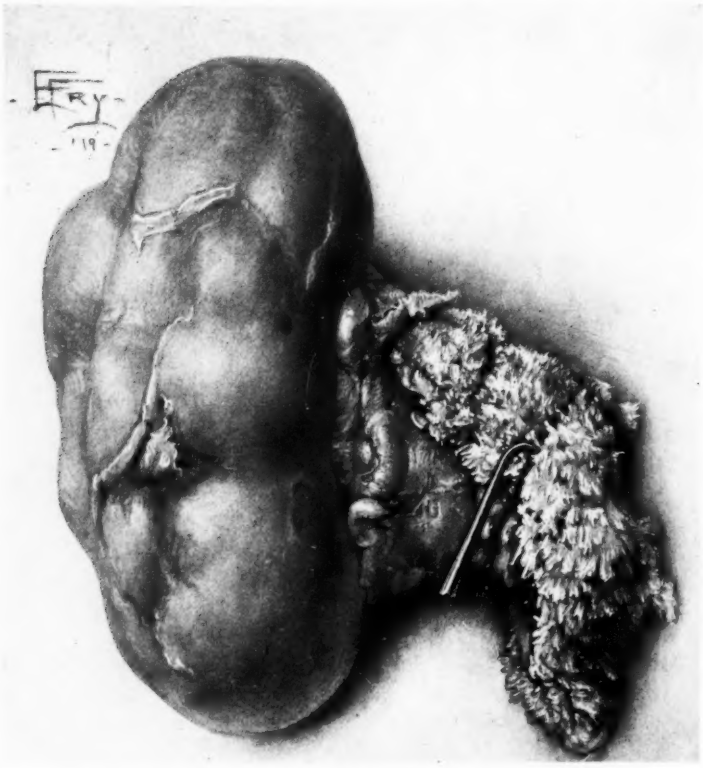

Fig. 15. Papillomatous masses protruding from the opened renal pelvis. (A212016.)

contained slight amounts of albumin and pus. The phenolsulphonephthalein test was 65 per cent. Cystoscopic examination, April 11, revealed a normal bladder. There was no secretion from the left ureteral orifice and the ureter was obstructed about 12 centimeters from the bladder.

April 12, 1919, through a left rectus extraperitoneal incision, the left ureter was found to be œdematous and attached to the surrounding tissues at about the brim of the bony pelvis. It was freed by dissection and an obstructing mass was felt. The ureter was opened and an organized blood clot was forced out by the intra-ureteral tension, followed by a pint of purulent urine. It was possible to pass a probe apparently into the pelvis of the kidney, and down as far as the bladder. The patient had an uneventful convalescence, leaving the hospital on the fourteenth day.

At cystoscopic examination May 13, the function of the left kidney was almost normal. A pyelogram revealed a few scattered dilated calyces, with a large, dimly outlined pelvis. The patient did not desire operative procedures at this time. March 8, 1920, he returned. He had almost constant hæmaturia since leaving the Clinic, and practically always a little blood in the urine, and sometimes clots. He had occasional attacks of pain in the left side. Urinalysis revealed both pus and blood in large amounts. The phenolsulphonephthalein test was 55 per cent. Cystoscopic examination March 10, revealed occasional spurts of hæmorrhagic urine from the left meatus and frequent spurts of clear urine from the right. There were several blood clots in the bladder. March 12, through a left lateral incision, a fixed hard kidney was removed with 8 centimeters of the ureter. Indurated tissue, which was found posteriorly, was not removed. Two clamps were left on the pedicle, on account of the friability of the tissue and the induration around the pedicle. The remaining portion of the ureter was ligated and dropped back into the wound. The clamps were removed on the fourth day. The patient convalesced uneventfully and left the hospital on the fourteenth day. September 12, 1922, the patient's general condition was excellent. There had been no recurrence.

The kidney weighed 490 grams. A large, papillomatous mass filled the pelvis and extended up into the renal tissue. The mass was covered with short, stubby protrusions, and extended down into the ureter blocking the pelvic outlet. Histologically the growth was composed of short papillæ which, through compression and fusion of adjacent fronds, had become matted together into a comparatively solid mass. The cells were irregularly situated around remnants of altered papillary tips. They were irregular in size and shape, and contained prominent nucleoli. There were a moderate number of mitotic figures. The growth was of moderate malignancy and was histologically similar to the flat, superficial type of papillary epithelioma of the bladder. The fact that there was no extension to the bladder from a kidney so extensively involved was possibly due to the obstruction at the ureteropelvic outlet.

Case 5. (A256716.) G. G., a man, age 34, came to the Clinic January 18, 1919, complaining of attacks of painless hæmaturia of 20 months' duration. Sixteen months before, he had had several attacks associated with pain on the left side, following which he passed a number of blood clots. For the last 9 months he had had almost constant hæmaturia. He was 20 pounds under normal weight, and complained of frequent headaches and rapid, forceful heart beats.

The urine contained large amounts of albumin and red blood cells, and a small amount of pus. The hæmoglobin was 45 per cent, the erythrocytes 2,540,000. Roentgenograms of the kidneys, ureters, and bladder were negative; the phenolsulphonephthalein test was 35 per cent. Cystoscopic examination revealed a small papilloma at the left orifice; this was destroyed by fulguration. The right orifice was normal. There was increased secretion from this side and a phenolsulphonephthalein return of 22 per cent in 15 minutes. The ureteral catheter encountered an impassable obstruction in the left ureter 7 centimeters from the bladder, and no urine was obtained. February 17, through a Mayo lateral incision, a lobulated kidney about twice normal size with a dilated pelvis, and the upper third of the ureter, were removed. The pelvis was found to be filled with a grossly malignant papillomatous mass which extended to the ureter. The patient was then turned on his back, and the ureter removed down to the bladder through a low, left rectus incision. The convalescence was uneventful and the patient left the hospital on the eleventh day. October 27, the patient returned for examination, although he had had no trouble. Cystoscopic examination revealed a recurrence in the left bladder wall, 3 centimeters in diameter. November 7, through a suprapubic incision a portion of the bladder, including the growth and the stump of the ureter, was removed. The patient convalesced from the operation readily, and left the hospital on the fourteenth day. May 4, 1920, he returned, complaining of frequency and burning at micturition. Cystoscopic examination

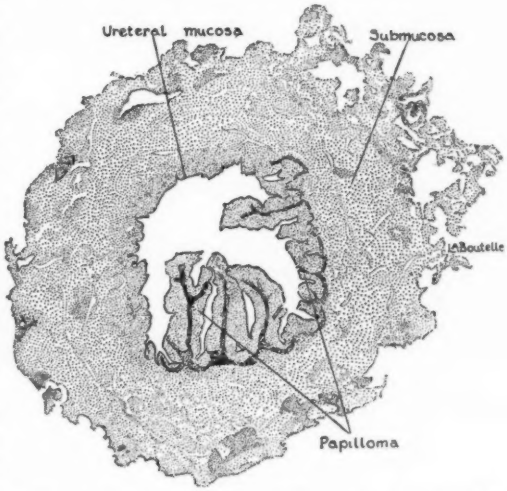

Fig. 16. Transplants from papilloma of the renal pelvis to the lower portion of the ureter.

revealed a flat, sessile mass, 2 centimeters in diameter, with a necrotic base, on the right wall. May 10, a suprapubic cystostomy was performed, and a 60 milligram radium capsule applied directly over the growth for 6 hours, being held in place by fine catgut. The remainder of the bladder was in good condition. For several months afterward the patient had a severe cystitis with swelling, tenderness, and a boggy œdema of the prostate. On subsequent cystoscopic examinations in 8, 11, 18, and 27 months, there was no evidence of recurrence, capacity of the bladder was normal and there was a definite area of scar formation where the radium had been applied.

The kidney was large and dilated, and most of the parenchyma atrophied or destroyed. The mass filling the pelvis was 6 centimeters in diameter and was composed of papillomata, some of fine texture, but the greater number short and stubby; they did not have the fine arborescence of similar tumors found in the bladder. The growth extended into the calyces and down into the ureter (Fig. 14). There were several papillomata from 1 to 3 centimeters in diameter scattered in the central portion of the ureter, which were grossly similar in appearance to those in the renal pelvis. In the bladder, the small, rounded, arborescent papillomata did not differ materially from the apparently benign growths usually found in the bladder. The tumors were loosely held together, most of them pedunculated, and the individual fronds were more wavy and arborescent than those of the kidney. Histologically, the renal mass was composed of papillomata of moderate length; fusion of adjacent papillæ was rare. The tumor cells in most areas conformed to

the usual benign appearance of a papilloma. In a few areas the cells were large, irregularly disposed, and contained prominent, deeply staining nucleoli. In the ureter there was more fusion and matting of the papillary masses, and in some areas of the connective tissue, supporting structures and blood vessels had disappeared. The cells were more irregularly disposed than were those in the renal mass. In some areas there was a slight cellular hyalinization, with a tendency toward epithelial pearl formation. In the bladder the papillæ were long and arborescent, and the individual cells small and regular in size and shape, but only rarely did they conform to the usual benign, transitional appearance of simple papillomata.

CASE 6. (A287879.) A. M. K., a man, age 49, came to the Clinic September 5, 1919, because of hæmaturia. Eleven months before, his urine had been tinged with blood for 4 days. Similar attacks had occurred 6 months and 3 months before, the latter lasting 3 weeks. Two months before, he had had a sharp attack of pain on the left side; this lasted 2 hours and terminated with the passage of a blood clot and a large quantity of bloody urine. He had had almost constant hæmaturia for 10 days, and recently frequency of micturition, nocturia and pyuria.

The physical examination was negative. The urine contained large amounts of pus and blood. The phenolsulphonephthalein test showed a 40 per cent return of the dye in 2 hours and 15 minutes. The hæmoglobin was normal, and roentgenographic examination of the urinary tract was negative. The left kidney was palpable. Cystoscopic examination revealed a normal bladder; both ureteral orifices appeared to be normal, clear urine spurting from both sides. On catheterization the urine from the right ureter was normal; that from the left was hæmorrhagic.

At operation, September 12, a normal sized kidney was exposed through a left Mayo lateral incision. A tumor was felt in the renal pelvis which extended into the upper pole of the kidney. The peritoneal cavity was opened and the opposite kidney explored and found to be normal. The left kidney was removed, together with 10 centimeters of the ureter, which was dilated and filled with blood clots. The patient convalesced uneventfully and left the hospital on the tenth day. September 3, 1920, he returned to the Clinic. He had gained in weight and his general condition was excellent. He had had moderate frequency and dysuria for several months, and two slight attacks of hæmaturia. Roentgenograms of the urinary tract were again negative. The renal functional test was 35 per cent; the urine contained yus and blood. Cystoscopic examination revealed a small papilloma, 2 by 1 centimeters, in the base of the bladder, and also one at the edge of the ureteral orifice. Both were completely fulgurated. April 18, 1921, the patient again returned. His general health was good and there had been no urinary symptoms except one attack of hæmaturia one week before. The urine still contained pus and blood and the functional test was 40 per cent. Cystoscopic examination revealed a smooth, meaty recurrent growth, 2 centimeters in diameter surrounding the left meatus. May 28, a left ureterectomy was performed and the tumor-bearing area in the bladder resected. The patient's convalescence was uneventful and he left the hospital on the nineteenth day with his wound healed. September 24, a cystoscopic examination revealed another recurrence 2 centimeters in diameter on the left wall. Four hundred milligram hours radium emanations in four tubes was planted directly into the tumor through the cystoscope. Several weeks later the growth was thoroughly fulgurated. December 27, cystoscopic examination revealed extensive radium cystitis, but no evidence of recurrence. May 1, 1922, the patient returned for observation. A small meaty circumscribed recurrent papilloma was found on the right wall. A 60 milligram radium capsule was applied to the growth for 1 hour. Later this area, and a small recurrent growth on the left wall were completely fulgurated. September 11, the patient returned complaining of slight frequency of micturition and moderate dysuria. His general health was good. Cystoscopic examination revealed a small papillomatous recurrence in the sphincter which, with two small areas in the posterior wall, was completely fulgurated.

The kidney was slightly larger than normal. The pelvis was dilated and thickened and contained a mass 4 centimeters in diameter in its upper half. It was somewhat hard and consisted of small, stubby papillomata matted together. The ureteral outlet was open and was not involved in the tumor. There was very little destruction of renal tissue, although the growth extended into the renal parenchyma at the upper pole of the kidney. The lower end of the ureter removed at the second operation contained several localized masses of flat papillomata, each about 2 centimeters in diameter, which encircled the wall in two places, filling and blocking the ureter. The upper end of the ureter near the point of ligation was thinned and dilated, probably from retained inflammatory products of the obstructed segment. At the insertion of the ureter into the bladder there was a mass 3 centimeters in diameter directly continuous with the ureteral growth. Histological sections from the kidney, ureter, and bladder are not essentially different in structure; they are composed of heavy papillomatous masses matted and fused together. In many areas the cells are regular in size and shape, having the usual palisade arrangement of bladder papillomata. A number of small, localized areas undergoing definite malignant change were found. In these areas the cells were large, irregular in size, deeply stained, and contained mitotic figures. Sections of the surrounding pelvic mucosa taken from uninvolved areas revealed a definite villous papillitis; in most sections the epithelial mucosa alone was involved in this process, but occasionally the submucous tissue also projected up into the small

villous irregularities. There was extensive round cell infiltration and moderate fibrosis in the deeper submucous tissues.

CASE 7. (A212016.) A. W., a man, age 55, came to the Clinic October 25, 1917, complaining of persistent hæmaturia of 9 months' duration. There had been no frequency nor dysuria until a few weeks before. Seven months before, at a cystoscopic examination, it had been found that the left ureter was partly obstructed and the urine from that side bloody.

General examination at the Clinic was negative. The urine contained pus and red blood cells in large amounts. The hæmoglobin was 65 per cent. The phenolsulphonephthalein return was 40 per cent in 2 hours and 15 minutes, and the blood urea was 27 milligrams for each 100 cubic centimeters of blood. Roentgenograms of the kidneys, ureters and bladder were negative. October 29, a cystoscopic examination revealed a normal bladder, containing clear urine. The right ureteral orifice was normal. The left was inflamed, and œdematous; it did not contract, and there was no secretion during 10 minutes' observation. The secretion from the right ureter was rapid and clear. Ureteral catheters were inserted into both ureters. A differential phenolsulphonephthalein test returned 20 per cent from the right ureter and nothing from the left in 15 minutes. A pyelogram of the left kidney revealed only a few irregular shadows.

November 17, the kidney was explored through a left rectus incision. This wound was closed, the patient turned on his right side, and a nephrectomy and partial ureterectomy performed through a left Mayo lateral incision. The ureter was very fragile, tearing readily; a section 10 centimeters in length was removed. The lower 5 centimeters was normal. The wound healed readily and the patient left the hospital on the fourteenth day. December 5, 1918, the patient returned to the Clinic. Four months before, he had passed blood once, and 1 month before he had passed several blood clots. The urine contained blood. Urination had been moderately frequent. The phenolsulphonephthalein return was 40 per cent in 2 hours and 15 minutes. December 7, cystoscopic examination revealed a growth at the site of the left ureteral orifice with an irregular cauliflower appearance. January 3, 1919, the bladder was opened through a suprapubic incision. A papilloma 3 centimeters in diameter projected from the left ureteral orifice and a smaller arborescent growth was found in the middle line posteriorly. A portion of the bladder, 6 centimeters in diameter, with the remaining segment of ureter, was removed. The peritoneum was opened and closed with plain catgut. The patient's convalescence was uneventful and he left the hospital with the wound closed on the twenty-fifth day. October 13, he returned, complaining of tenderness in the inguinal region and of frequency of urination. November 1, cystoscopic examination revealed multiple malignant papillomata in the base of the bladder. The patient was given 3,320 milligram hours of radium (2,800 milligram hours in a suprapubic pack, 320 milligram hours rectally, and 200 milligram hours direct to the growth through the cystoscope), and allowed to go home. Three months later he returned, complaining of frequency and urgency. Cystoscopic examination, February 9, 1920, revealed that the tumors seen at the last examination had increased markedly in size. March 24, a third operation was performed, the growths in the bladder being excised by cautery. The patient left the hospital with the bladder wound healed on the twenty-fourth day after operation. Cystoscopic examination 6 weeks after the operation did not reveal evidence of malignancy in the bladder. April 4, 1920, three small recurrent growths were removed by fulguration. January 28, 1921, cystoscopic examination revealed multiple recurrent growths in the base of the bladder. The growth was rather inaccessible and the patient intolerant to fulguration. The general condition and renal function were excellent. February 4, 1921, the fourth operation was performed. The bladder was opened through a suprapubic incision and multiple growths were excised and their bases cauterized. It was also necessary to remove the prostate in order to reach the prostatic urethra, which was involved in the growth. The patient's convalescence was fairly normal until the twentieth day, when he became uræmic. He died March 8.

Necropsy revealed marked right pyenephritis in an extremely large dilated kidney. There were also fatty changes in the liver and myocardial degeneration. The right kidney was small, and its pelvis markedly dilated and thickened. Only a small amount of kidney tissue remained. The pelvis and calyces, also markedly dilated, were filled with short, stubby, papillomatous masses extending into and almost completely blocking the ureter (Figs. 15 and 16). The pelvis between the areas of growth was thickened, rough, and occasionally fibrosed. The wall of the ureter was thickened. The upper 5 centimeters was almost completely covered by short, stubby papillomatous protrusions, similar to those of the renal pelvis. The growths removed from the bladder at the last three operations were similar. They varied in size from 1 to 4 centimeters in diameter, were looser, and the villi were long and freer than those of either the pelvis or the ureter. The renal mass was composed of fronds of moderate length, with the palisade arrangement of benign papillomata. Fusion of adjacent fronds was common. The supporting connective axis and the central blood vessels of individual fronds, or of small groups of fronds came directly from the underlying submucosa. There was no extensive pedunculation. The epithelial coverings of the papillæ were readily traced as a continuation and proliferation of the normal transitional mucosa. There was moderate œdema in the submucosa, but no evidence of invasion by the tumor. In the ureter the fronds were short and stubby; in many areas they were completely fused with large cell masses. There was no

free, wavy branching. In some areas the central supporting axis had completely disappeared, the growth having lost its papillary structure. There was extensive round cell infiltration, fibrosis, and thickening of the submucosa. In the bladder the fronds were much longer and thinner than those of either the kidney or ureter, having the usual wavy arborescence seen in benign or moderately malignant papillomata of the bladder. In certain areas the regularity of the palisade arrangement had been lost, the individual cells being irregularly disposed and staining deeply. In a few areas there was matting together of individual fronds with loss of papillary formation, similar to that occurring in the kidney. The adjacent mucosa of the bladder was distinctly thickened, forming papillary irregularities which gradually tapered off into normal mucosa. Sections from the three organs indicated a moderately malignant process.

CASE 8. (A34504.) Mr. A. W., a man, age 47, was examined in the Clinic February 21, 1910. For 30 years he had had attacks of right renal colic at intervals of from several months to several years. Between attacks he was generally quite well. For the past 2 years the attacks had been more frequent, and for the last year pain had been almost constant in the right renal area. The patient had lost 40 pounds in weight and his urine was tinged with blood most of the time. Physical examination revealed a cystic mass in the upper right abdomen. The urine contained albumin, pus and blood. The hæmoglobin was 70 per cent. A roentgenogram of the urinary tract revealed several small shadows in the right renal area. Cystoscopic examination revealed normal bladder mucosa. The left ureteral opening was normal in appearance. The right orifice was eroded and spurting cloudy urine. Catheters were passed into both ureters; the urine from the right contained pus; from the left, it was normal.

March 1, 1910, a right nephrectomy was performed through a lateral incision. The renal mass was so large that it was necessary to break the twelfth rib to remove it. Two pairs of clamps were left on the pedicle; these were removed on the third day. The patient's convalescence was uneventful, but he died suddenly 5 months later.

The kidney was tremendously dilated, with a thinned out pelvic wall. All the parenchymatous tissue had completely atrophied, and the pelvis and calyces were filled with a soft, tumorous mass, covered with short, irregular, papillomatous protrusions. The ureteral outlet was filled with the growth. Several stones, 1 to 2 centimeters in diameter, were found free in the pelvis. The tumor consisted mainly of actively malignant cells arranged in a papillomatous formation around the central axis of connective tissue. The cells were large and irregular with clear outlines. In some areas individual papillæ with a regular arrangement of cells indicated that this tumor belonged primarily to the group of papillary epitheliomata. In other areas the central connective tissue stalk was retained, but the cells were larger, more irregularly disposed, with an increased amount of protoplasm and large, deeply staining, prominent nuclei. The cells in this area resembled the papillary adenocarcinoma type, which is not infrequently found arising in the parenchymatous portions of the kidney.

SUMMARY

The majority of tumors of the renal pelvis are papillomatous. Various irritative conditions, infection and stone are predisposing factors in the development of these tumors. The growths may be small, pedunculated, and grossly benign, or they may completely fill the renal pelvis, blocking the ureteral outlet, and by obstruction and invasion convert the kidney into a functionless dilated sac.

On cystoscopic examination the bladder may appear normal. Small papillomatous transplants may be found protruding from, or surrounding the ureteral orifice. In some cases multiple small transplants are scattered extensively over the mucosa of the bladder.

The papillary growths in the renal pelvis, as well as those in the ureter, are more compact than the transplants to the urinary bladder. As in most papillomatous tumors of the bladder, numerous areas in the growths in the renal pelvis are undergoing malignancy. The majority of tumors of the renal pelvis are malignant histologically. Clinically the transplants to the lower urinary tract, the extension to neighboring tissues, and the local recurrences, make these tumors all potentially malignant.

Because of the frequency with which the ureter is involved, and the repeated recurrences after nephrectomy, a complete nephroureterectomy is essential to insure even partial success.

There were 8 papillary tumors of the renal pelvis in the series; all were histologically malignant. Three patients died from 5 to 9 months after the operation, one of whom had a transplant to the ureter, and another, a large secondary growth in the bladder. A fourth patient died from uræmia 4 years after a nephrectomy. During these 4 years, the patient had repeated multiple transplants which were treated by removing the ureter, by resecting the bladder, and by extensive fulguration. Four patients are still alive, two are

free from recurrence of transplantation of the growth, one 2½ years, and the other, 4 months after the removal of the diseased kidney and ureter. The remaining two patients have had repeated transplants to the bladder, requiring persistent treatments; at the present time both patients are well, one, 2 years, the other, 3 years after the first operation.

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INTRACRANIAL HÆMORRHAGE IN THE NEWBORN[1]

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INTRACRANIAL hæmorrhage of the newborn is a condition which is mentioned in most of the modern textbooks but few pages are devoted to the detailed discussion of the subject. This malady is of far greater occurrence than is commonly believed. Holt (1), quoting Cruveilhier, states that at least one-third of the deaths of infants which occur during parturition are due to meningeal hæmorrhage.

In the past few years more interest has been evinced in this subject as is shown by the increased number of papers published (Greene, 2; Brady, 3; Sidbury, 4). In a fairly recent contribution to this subject Warwick's (5) important summary of a necropsy report on hæmorrhage in the newborn is as follows:

1. "Cerebral hæmorrhage of the newborn is frequently found, occurring in 50 per cent of the 36 fatal cases of young infants at the University Hospital.

2. "The condition is brought about by trauma in normal or rapid deliveries, by congestion or asphyxiation in slow deliveries, or by disease of the child itself.

3. "Hæmorrhagic disease of the newborn is a very important cause of cerebral hæmorrhage in infants, occurring in 44 per cent of the deaths in our series."

In our series of 100 consecutive deliveries there was only one case which the pediatrician clinically diagnosed as hæmorrhagic disease of the newborn. This happened in a luetic child with a 4 plus Wassermann reaction who began to have hæmatemesis and melæna on the second day and died within the next 24 hours. Two lumbar punctures, one 12 hours and the other 36 hours after birth, revealed clear spinal fluid under normal pressure. The coagulation time was 4½ minutes done on the first day of life. Rodda (6), however, states that the coagulation time usually begins to lengthen on the third day of life, though it may begin at the sixth hour. At postmortem examination intracranial hæmorrhage was absent.

4. "Forcep deliveries, advanced age of the primipara mothers and syphilis probably do not play as important a rôle in the etiology of this condition as was formerly supposed."

In a still more recent article Huenekens (7), makes the emphatic statement that the recognition of cerebral hæmorrhage of the newborn is a most neglected phase of the care of the newborn while it is a most important one.

Sharpe and Espejo (8) in a series of lumbar punctures on 100 consecutive newborn babies within the first 48 hours ascertained intracranial hæmorrhage in 9 per cent. All these occurred in normal, or so-called normal, deliveries.

PATHOLOGY

The size of the extravasation of blood may be small or large, varying in amount from 1 dram to 2 ounces or more. The small hæmorrhages may be single or multiple and they are usually all subdural and subarachnoid anatomically. In addition to being supracortical the large hæmorrhages may also occur intraventricularly. Cerebral œdema often accompanies intracranial hæmorrhage. Uncomplicated extradural hæmorrhage is rarely seen. To quote Holt (1): "Extradural hæmorrhage may be said never to occur except when associated with fracture."

The very small and signless hæmorrhages are rarely sufficient to cause death and may in our opinion be absorbed by the natural processes without producing any resultant defect in the child's future normality. Holt (1), on the other hand, claims that even small hæmorrhages usually cause some permanent injury though this may not be manifested for years. Other hæmorrhages may be of such a size as not to cause death, yet, after absorption of the blood, do leave behind them an organization residue coating the supracortical vessels and especially the veins which constitute approximately 80 per cent of the normal channels of excretion of

[1] Delivered at the New York Obstetrical Society, October 9, 1923.

the spinal fluid. The resulting situation would necessarily produce an external hydrocephalus of varying degree followed in the future by some secondary cerebral impairment or spastic condition or both. If the organized tissue sealed the foramina of Magendie and Luschka, then, in addition to the external, an internal hydrocephalus would be produced.

These cases eventually grow up to be known as Little's disease though cerebral spastic paralysis may be caused by cerebral agenesis and meningoencephalitis. Little (9) in 1843 stated that cerebral hæmorrhage in the production of cerebral spastic paralysis was the factor in only a few instances. Nineteen years later (1862) he (10) placed the figures of hæmorrhage as the cause to be as high as 75 per cent. Spastic paralysis, whether it is of the diplegic, paraplegic, or hemiplegic type caused by intracranial hæmorrhage at birth, should be excluded from the group of true Little's disease; the latter should include only the agenetic and meningoencephalitic forms for the reason that the intracranial hæmorrhage cases are amenable to drainage if seen early enough in the initial stage.

SYMPTOMATOLOGY

The cases are brought to the neurologists or neurosurgeons late when the mothers notice that their children fail to sit up or walk, teething is delayed, mental impairment is present, paralysis is manifest, or some such obvious sign is present. If we are more fortunate, the cases are seen early. However, in such instances the conditions are severe enough to produce extreme stupor, cyanosis, repeated convulsions, spastic paralysis, opisthotonus, bulging anterior fontanelle, and the separation of sutures. As a rule the hæmorrhage is of necessity large in these cases. The more common situation, which is for the most part overlooked and permitted to remain undiagnosed, are the extremely mild cases wherein slight drowsiness, apathy, few muscular twitches, failure to nurse properly and feeble or irregular respirations constitute the clinical picture, or even these in the mildest cases may be entirely wanting. Holt (1) states that cerebral hæmorrhages are frequently found when there have been no signs referable to the brain and that it is a question whether they are not quite a common sequel of labor. Holt is aware of the frequency of the condition and he belongs to the minority group.

OCCURRENCE

With this in mind, this present investigation was carried on as a continuance of the first series of 100 cases to determine, if possible, what percentage of newborn babies sustain intracranial hæmorrhage and cerebral œdema, with or without signs, and whether a routine method could be adopted whereby cerebral vascular injury without signs could be detected early. This investigation will be continued until a larger series has been studied, and under the same existing conditions.

Permission was obtained to continue this line of research on the maternity division at the City Hospital, Welfare Island, through the courtesy of Drs. F. A. Dorman and Wilbur Ward.

Routine lumbar punctures were performed on the second 100 consecutive newborn babies, ranging in age from 9 minutes to 100 hours. The intention was to do all these punctures within the first 24 to 48 hours and only in 7 cases were the babies older. In one of these, a 53 hour old baby, was bloody spinal fluid present. The 2 infants, one 9 and the other 14 minutes old, had clear spinal fluids; the former had an intracranial pressure of 12 millimeters mercury, and the latter a pressure of 4 millimeters mercury. The normal intracranial pressure as registered by the spinal mercurial manometer at lumbar puncture is 4 to 8 millimeters mercury. It is, however, advisable to delay performing this test until 12 hours after birth to permit the child to adapt itself to the new environment and to adjust its mechanism to its new surroundings.

The technique consisted of the following: an intramuscular hypodermic needle was used instead of the regular lumbar puncture needle. The spinal puncture was done in the fourth lumbar interspace. The child's back was well flexed by a nurse. After the needle entered the spinal canal and spinal fluid was obtained, the child was relaxed from its strained position and as a rule ceased crying.

With the child quiet the intraspinal manometric reading was taken. If blood tinged, or bloody spinal fluid was obtained, another puncture, one space higher, was made. If the second puncture, performed immediately after the first, was clear, then the tap was considered a clear one; if, however, the character of the fluid was the same as in the puncture below, then that index was taken. To consider a fluid blood-tinged, or bloody, the spinal fluid had to be well mixed with blood and not blood-streaked, also several cubic centimeters of fluid of the same consistency had to be removed. Twenty-four hours later, a second drainage tap was performed in the bloody cases and in those with cerebral œdema, the former for the purpose of removing the hæmorrhage, and in the latter to reduce the œdema and the intracranial pressure. In the bloody cases the spinal fluid obtained on the second puncture varied in concentration of blood from straw to cherry red depending upon the character of the spinal fluid and the quantity of hæmorrhage present in the first puncture. As a rule, the spinal fluid appeared clearer at each successive lumbar puncture. The third drainage puncture was performed 48 hours after the first puncture, and the spinal fluid continued to show either a diminution in the blood concentration, or a restitution to the normal colorless spinal fluid proving that the hæmorrhage had been entirely drained. Some, of course, was absorbed. If three punctures failed to suffice to drain the hemorrhage as many more were performed at 24 hour intervals as were necessary to procure clear spinal fluid. The test tubes containing the bloody fluid were left to stand for 30 minutes to determine the presence or absence of coagulation. Bloody spinal fluid does not clot readily whereas pure blood does. In this manner an intravenous puncture of the spinal plexus of veins was differentiated from true bloody spinal fluid. In addition, in questionable cases it is always possible to resort to the Benedict test for sugar; the latter being constantly present in the normal spinal fluid in sufficient amount to give the reaction, whereas the reduction in the blood is not found.

FINDINGS

In 13 cases of 100 consecutive newborn babies, bloody spinal fluid of varying degree was found. The fluid ranged in color from straw (in a 37 hour old baby) to bright cherry red.

In the blood-tinged, or bloody spinal fluid, repeated lumbar punctures every 24 hours were performed until the fluid was clear. In only 3 cases was this not accomplished. One case, a medium forceps, the second of a pair of twins, at the first puncture had a manometric reading of 8 millimeters mercury, six cubic centimeters of bloody spinal fluid was removed the first day; four more punctures were performed at 24 hour intervals and only 1 or 2 cubic centimeters was obtained at each tap. The pressure was not taken at the subsequent taps, and the child left the hospital with the spinal fluid still blood-tinged. The second case was the child of a 4 plus syphilitic woman who had been in labor 5 days and finally delivered as a breech. The initial spinal pressure was 6 millimeters mercury; 5 cubic centimeters of bloody spinal fluid was removed the first day; two more taps were made 24 and 48 hours later when only a few drops of bloody fluid escaped. This case also never had a clear fluid during its hospital residence. The third case was that of a face presentation. The first manometric reading was 4 millimeters mercury; only 1 cubic centimeter of bloody fluid was obtained on lumbar puncture. Another puncture, 36 hours later, yielded a few drops of spinal fluid which was less bloody than the initial tap showing that absorption had well progressed. The hæmorrhage probably was extremely small in this latter case. The second case showed twitches of the hands and the third case had twitches of the face, mouth, hands, and feet, also cyanosis of the face. The twitches ceased after the initial tap in the second case, and on the fourth day in the third case.

In four cases, one puncture was sufficient to drain the hæmorrhage in that the second puncture was clear. In two cases two punctures were required; in one case three punctures were necessary; in another, four punctures were performed and in still another five

TABLE I.—ONE HUNDRED CASES—CONSECUTIVE DELIVERIES

	Cases	Total
Males	55	
Females	45	100
Primiparæ	51	
Multiparæ	49	100
Clear spinal fluid	81	
Blood-tinged or bloody	13	
Canals not entered	6	100

punctures. One case, that of a 7 month premature baby weighing 2 pounds and 12 ounces was punctured 15 minutes after death; its fluid was blood-tinged. The autopsy was delayed several days and careful examination of the brain was not obtained.

The intracranial pressure in the 13 bloody cases ranged from 4 to 26 millimeters mercury and was as follows: 4 millimeters mercury in five cases, 6 millimeters mercury in one case, 8 millimeters mercury in three cases, 10 millimeters mercury in two cases, 12 millimeters mercury in one case and 26 millimeters mercury in another. The blood-clotting time was estimated in 9 within the first 24 hours, 2 between 24 and 48 hours, 1 at 53 hours of life and one 15 minutes after death, and in these 13 cases it was as follows: 7 minutes in 1 case, 7½ minutes in 2 cases and in the other 10 cases, the time ranged between 4 to 6½ minutes, the normal being 5 to 8 minutes. In one of these cases, the mother had a 4 plus blood Wassermann reaction, while the child's umbilical cord Wassermann was negative. In another the mother had a 1 plus blood Wassermann and the child was negative. Of these 13 bloody cases, 6 were normal cephalic deliveries, 3 prolonged labors, 2 of which terminated as cephalic presentations, and 1 as a breech, 2 medium forcep deliveries, one of them the second of a pair of twins, one a face presentation and one the child of an eclamptic mother. The anterior fontanelle in these 13 bloody cases was flush in 7 cases, bulging in 3, depressed in 1, and not recorded in 2. The anterior fontanelle was in no way indicative of the spinal pressure obtained. Only two of these babies had the mildest of signs; namely, twitchings of the face, hands or feet and cyanosis of the face. None of the marked signs, such as convulsions, stupor, spasticity, failure to nurse, etc. were present.

TABLE II.—THIRTEEN BLOODY CASES

Nos.	Name	Age	Character of spinal fluid	Spinal fluid pressure millimeters mercury	Clotting time	No. of punctures
1	F. D.	37 h.	Straw	4	4 min.	1
2	J. C.	20 h.	Blood-tinged	4	6 min.	1
3	P. C.	15m.A.D.	Blood-tinged	4	none	done
4	M. L.	19 h.	Blood-tinged	4	7 min.	1
5	S.T.H.	3¾ h.	Bloody	26	4 min.	3
6	R. N.	53 h.	Bloody	12	7 min.	1
7	N. E.	10½ h.	Bloody	10	8 min.	5
8	H. K.	35½ h.	Bloody	8	6½ min.	2
*9	P. T.	4 h.	Bloody	8	7½ min.	*5
10	A. G.	6 h.	Bloody	10	5 min.	2
*11	W. B.	15 h.	Bloody	6	7½ min.	*3
*12	A. B.	12 h.	Bloody	4	5½ min.	*2
13	A. I.	22 h.	Bloody	8	4½ min.	5

*Left hospital—fluid not clear.

Eight were first borns, 3 the second, 1 the seventh, and 1 the twelfth. Twelve were males and 1 was a female.

OTHER OBSERVATIONS

One baby was jaundiced and died. The spinal fluid was clear and the coagulation time was 7 minutes, determined 14½ hours after birth. The necropsy failed to include the cranium. Another case, which was a medium forceps delivery, had bloody spinal fluid and developed jaundice on the third day with a temperature of 102 degrees F. The blood clotting time lengthened from 4½ minutes to 6 minutes. Four lumbar punctures were necessary to obtain clear spinal fluid. Further study must be made of the relationship, if any, between intracranial hæmorrhage and jaundice and the allied icteroid conditions; whether the absorption of hæmoglobin of an intracranial hæmorrhage can produce the clinical picture of a mild degree of icterus has not been definitely proven.

Ten babies of this series were of luetic mothers, 2 of them had an intracranial hæmorrhage, while in the remaining 8 the spinal fluid was clear. Of these luetic children the intracranial pressure was normal in 7, in one it was 10 millimeters mercury, and in two it was 12 and 14 millimeters mercury,

TABLE III.—FORCEPS CASES

	Low forceps	Medium forceps
Number of cases	8	2
Clear spinal fluid	6	0
Bloody or blood-tinged	0	2
Canal not entered	2	0

TABLE IV.—BABIES OF SYPHILITIC MOTHERS

	Cases	Total
Clear spinal fluid	8	
Bloody or blood-tinged spinal fluid	2	10
Normal spinal pressure	7	
Increased spinal pressure	3	10

respectively. Signs of twitchings, refusal to nurse or difficulty in suckling and cyanosis were exhibited in 8 cases, of these one had a spinal pressure of 14 millimeters mercury, five were normal and in two the punctures were not successful due probably, to faulty technique, or to anatomical variations of the spinous processes or of the bony canal itself. Ordinarily a "dry tap" occurs only in cases of pyogenic meningitis where the pus is too thick to flow out through the needle and where the trap door of the dura formed by the entering needle may clog the lumen. Cerebral œdema of varying degree without hæmorrhage or signs as determined by the spinal manometer was present in 16 cases. The mothers of two children had an active gonorrhœa. One of the former had a doubtful Wassermann reaction (1 plus) and that mother's child had bloody spinal fluid. This series contained 51 primiparæ. Fifty-five of the babies were males. No untoward signs developed as a result of the puncture in this series.

Let us digress a moment to narrate a case seen in private consultation, and which left a marked impression upon us as well as to cause us to modify the original outline of treatment.

The writers were called, one afternoon, August 28, 1923, to see a patient delivered by Dr. S. Swift. The case was a very difficult forceps delivery, and the baby weighed 9½ pounds. The right parietal region was flattened while the left corresponding side gave evidence of a cephalhæmatoma. That same afternoon a lumbar puncture was performed, the baby being then 5½ hours old. Shock was present as indicated by the slight cyanosis, the cold skin and slightly labored

TABLE V.—CHARACTER OF SPINAL FLUID IN CASES OF PROLONGED LABOR

	Cases	Total
Bloody spinal fluid	3	
Clear spinal fluid	2	
Canal not entered	1	6

TABLE VI.—CHILDREN WITH SIGNS AND THE RELATIONSHIP OF THE ANTERIOR FONTANELLE AND SPINAL PRESSURE

	Cases	Total
Twitches of hands, legs, face, and mouth, cyanosis, poor nursing—7 cases.		
Bloody spinal fluid	2	
Clear spinal fluid	4	
Canal not entered	1	7
Anterior Fontanelle		
Bulging	1	
Flush	3	
Depressed	1	
Not recorded	2	7
Spinal Fluid Pressure		
Normal	5	
Increased	1	
Not noted	1	7

TABLE VII.—FACE PRESENTATION CASES AND THEIR SPINAL FLUID

	Cases	Total
Bloody spinal fluid	1	
Clear spinal fluid	1	
Canal not entered	1	3

respirations. A lumbar puncture revealed bloody spinal fluid under a pressure of only 3 millimeters mercury and 5 cubic centimeters was withdrawn. Three hours later, the child died (8½ hours after birth). That same evening an autopsy was performed; a hæmorrhage of approximately 12 ounces was found within the scalp tissues on the left side; a fracture depression of the entire right parietal bone was present and all the cerebral sulci contained bright red blood. No large supracortical clot was observed. The ventricles were clear.

The fact that the condition of shock in this child became worse following the lumbar puncture at which the bloody spinal fluid was ascertained under a pressure of only 3 millimeters mercury would tend to indicate that in cases of shock, just as in adults having acute brain injuries, no examinations, manipulations, or special tests should be performed until the acute stage of shock has subsided for fear of increasing the shock, thereby lessening the patient's chances for recovery for life.

There were 10 forceps deliveries; of these 8 were low forceps and in 7 the spinal fluid was clear and one canal was not entered; two were medium forceps cases both of which had bloody spinal fluids.

ETIOLOGY

In these cases of intracranial hæmorrhage where the labor apparently has been normal, where extraneous factors such as forceps or pituitrin (the latter was not used in any case in this series) were absent, in fact where the physician played a minor or secondary part in the delivery, it seems that the most important etiological factor in the production of cerebral vascular trauma may be attributed to rupture of the supracortical venous tributaries as they enter the sinuses whether due primarily to the overriding of the cranial bones caused by the tremendous pressure to which the head is subjected in the normal process of molding, or to an extreme congestion and dilatation of the supracortical veins.

Hæmorrhagic disease of the newborn probably does not play so much importance as a factor in the etiology as formerly believed. It is interesting to note that the coagulation time is ordinarily found lengthened only upon and after the third day although it rarely may be present as early as the sixth hour. In these series the clotting time has been estimated within 24 to 48 hours after birth and thereby explains the fact that in no case of intracranial hæmorrhage of the newborn was the coagulation time prolonged. In this series, one case clinically diagnosed as hæmorrhagic disease in the newborn did not have an intracranial hæmorrhage and the other 12 cases having bloody fluids did not have the disease.

Forceps as a factor becomes more important where the application is a late difficult one, or where it is a medium or especially a high, rather than a low forceps.

TREATMENT

Holt (1) advises surgical intervention early as he doubts the value of lumbar punctures.

Our experience in these cases can be summarized by the following; if death from an intracranial hæmorrhage occurs on the first or second day the blood is found partly fluid and partly clotted; this early clotting of the blood may be entirely postmortem or may occur during the moribund period since cranial operations as late as the fifth day after birth have revealed no clotting of the intracranial hæmorrhage. If death occurs on the fourth or fifth day the blood may be entirely coagulated and may begin to undergo partial absorption. Therefore, if lumbar punctures are performed before the blood has had an opportunity to begin to clot, the repeated spinal drainage will draw off a certain amount of blood at each tapping, the quantity remaining will mix with the additional new cerebrospinal fluid secreted and the blood dilution will be increased. This will tend to prevent coagulation. If, however, after repeated lumbar punctures the blood concentration of the spinal fluid shows no tendency to diminish, then we firmly believe that a modification of the subtemporal decompression and cranial drainage as described by Cushing is indicated in order to prevent impairment of the child's future normal development. The early use of calcium lactate or blood serum to aid in lessening the amount of the intracranial hæmorrhage may be of use. An important point always to be remembered is not to perform any tests whatsoever if the infant is in shock. This fact cannot be too strongly emphasized. Cases that are in shock should be treated as one of us (11) has outlined for the acute traumatic brain injuries in adults. Therefore, in these cases of intracranial hæmorrhage in the newborn in the presence of shock, recovery from shock is of primary importance; the spinal drainage of repeated lumbar punctures may then be used, and if that fails then a modified subtemporal decompression and cranial drainage.

SUMMARY

As intracranial hæmorrhage in the newborn occurs more frequently than formerly believed, every child evincing the mildest signs of cerebral irritation, or of increased intracranial pressure, should be subjected to a lumbar puncture. The performance of a lumbar puncture in experienced hands is a

safe procedure, and the child does not suffer any ill effects from it with the one exception stated above with the child being in severe shock. In addition, the bloody cerebrospinal fluid at birth is in a fluid state so that it can be easily drained by repeated lumbar taps, and the more major procedure of a modified subtemporal decompression relegated to those cases wherein lumbar drainage fails, and in this manner the institution of early therapy will tend to prevent many pitiful and helpless cases of cerebral spastic paralysis.[1]

CONCLUSIONS

1. Intracranial hæmorrhage of the newborn occurs more often than formerly suspected.

2. Death results from extensive intracranial hæmorrhages and cerebral œdema unless the hæmorrhage can be entirely absorbed by the natural means of excretion without any resultant organization residue; the unrecognized and therefore improperly treated cases of the milder degree of intracranial hæmorrhage develop in a large percentage later some form of cerebral spastic paralysis with or without mental impairment.

3. Cerebral spastic paralysis due to intracranial hæmorrhage should be differentiated from Little's disease—the latter including only those cases due to cerebral agenesis and meningo-encephalitis.

4. Pediatricians, neurologists, and neurosurgeons usually see the cases late as chronic conditions when spastic paralysis in its various forms has already developed, and when the condition at best can only be improved, whereas it is the obstetrician who sees these cases in the acute stage.

5. The acute cases with mild signs or no recognized signs at all are overlooked.

6. A study was undertaken to detect apparently signless intracranial hæmorrhage in the newborn with the result that 13 per cent of 100 consecutive deliveries at the City Hospital were discovered as having an intracranial hæmorrhage of varying degree by routine lumbar puncture within the first 24 to 48 hours after birth.

[1] It is most advisable and our intention to examine these patients having had an intracranial hæmorrhage at the time of birth at frequent intervals over a period of years.

7. Low forceps and syphilis were not found to be important factors in the causation of intracranial hæmorrhage at birth.

8. Lessened coagulability of the blood does not appear to be an important and frequent factor in intracranial hæmorrhage of the newborn—the clotting time not being lengthened in any of the cases in this series within 48 hours after birth.

9. Lumbar puncture as a diagnostic and therapeutic measure has proven to be a safe procedure in this series in the absence of shock.

10. Apparent signs indicative of an acute intracranial hæmorrhage and cerbreal œdema can be confirmed or disproven by early lumbar puncture and the resulting intracranial pressure estimated with the spinal mercurial manometer, and in doubtful cases the appropriate treatment instituted early.

11. Lumbar puncture is advocated as a safe routine procedure in suspected cases having the mildest signs of intracranial hæmorrhage and cerebral œdema within 72 hours after birth and is a valuable aid to the natural means of their absorption.

12. Repeated spinal drainage by means of lumbar puncture at intervals of 6 to 24 hours is advocated in cases of bloody spinal fluid under varying degrees of pressure.

13. If repeated lumbar drainage fails to diminish progressively the blood concentration and the pressure of the cerebrospinal fluid, then a modified subtemporal decompression and cranial drainage is indicated.

14. The fact that an intracranial hæmorrhage occurred in 9 per cent in the first series of 100 consecutive deliveries and in 13 per cent in the second series of 100 would tend to indicate a more frequent intracranial lesion at the time at birth than ever conceived.

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A SYMPOSIUM ON PAIN

THE SURGICAL SIGNIFICANCE OF PAIN[1]

By WILLIAM D. HAGGARD, M.D., F.A.C.S., Nashville

"There is purpose in pain,
Otherwise it were devilish."
—Owen Meredith.

PAIN is the chief defense mechanism against injury. It apprises us of many diseased states and accidents. It has been spoken of as the language of disease, but it is often particularly meager, many times greatly involved, frequently misleading, and sometimes perplexingly silent. Pain in some regions is easily recognized as characteristic of definite pathological processes. It is ofttimes bizarre and mixed with many conflicting manifestations. Moreover, it is so greatly modified by the individual as to be deceptive. The hyposensitive type of individual will endure severe pain with little outcry. A highly neurotic subject becomes an amplifier. Ordinary pain in them is increased to the *n*th power and makes them the subject of the surgeon's greatest solicitude. The phlegmatic are notoriously uncomplaining. The stoic minimizes pain that is of serious import. The neuropath in acute lesions suffers great agony and in chronic disease, though he may not suffer, his family suffers.

The cerebrospinal system, a later addition to the nervous system, is the real outpost against injury. The vegetative nervous system which has to do with the primitive processes presides over these essential functions and at times conveys pain in an exaggerated way during the normal events of digestion and elimination. If harmful afferent stimuli pass to the cord over a long period of time, the threshhold of response of the nerve cells which receive these stimuli is necessarily lowered and they respond to a stimulus which is much below that which they would ordinarily withstand. Hyperirritability of these cells permits a lesser stimulus than normal to produce a heightened response.

A patient whose sympathetic system is out of tune and who has so-called "nervous indigestion" will complain more bitterly than one who has a real pathological entity like an ulcer of the stomach. The triad of hunger pain, food ease, and night pain, relieved by vomiting or alkalis, made Moynihan say that the diagnosis of duodenal ulcer could be made by correspondence. While nothing is more telltale than the explosive upper abdominal pain that goes through to the back in gallstone colic, at the same time a considerable proportion of cases of gall-bladder disease do not have this frank manifestation. Subscapular pain is referred by the way of the sympathetic through its connection with the fifth or sixth dorsal nerve.

The complications of most diseases obscure the initial pain, but fortunately tenderness, rigidity, and temperature complete the syndrome of infection. Acute perforation of the hollow viscera is manifested by primary, sharp, stabbing abdominal pain quickly followed by the excruciating pain of intense peritonitis.

Acute perforation of the stomach and duodenum is frequently attended with collapse and followed by localization of the infectious material in the right iliac fossa, so frequently that perhaps one-third of the cases of perforation of the duodenum are diagnosed as appendicitis.

The complication of gall stones when the pancreas is infected is denoted by the most severe abdominal pain associated with vomiting and collapse, followed in 24 hours by an elastic, fluctuant, epigastric tumor. At first it is often regarded as acute intestinal obstruction to be later recognized as acute hæmorrhagic pancreatitis, the most dramatic catastrophe in the abdomen.

The well known epigastric pain of appendicitis, localizing itself in the right iliac fossa,

[1] Introduction to the symposium on pain presented before the Clinical Congress of the American College of Surgeons, Chicago, October 22–26, 1923.

is very uniform and familiar when the appendix is in its usual position to the inner side of the cæcum. The symptoms are usually so frank that the diagnosis is easily made. When the appendix is behind the caput, the mild unreferred pain will not challenge recognition and will allow grave suppuration to supervene before it is detected.

Perhaps the most deceptive pain in the abdomen to the surgeon is abdominal pain in children with beginning pneumonia where the diaphragmatic pleura is involved and mimics appendicitis. This should cause the clinician to be constantly on the alert for this dissimulation and make the wary examine the chest with great assiduity.

The very severe pain in the trajectory of the lower dorsal nerves extending around the right costal arch, may, in herpes zoster, simulate a mild cholecystitis and the true cause will be discerned only when the vesicular pattern appears on the third or fourth day.

We have learned to look upon abdominal pain with such scrutiny and realize so well its portent that I have seen a diagnosis of intestinal obstruction made from the pain and allied symptoms alone when the inguinal hernia that was the cause of the obstruction had never been observed. While pain is a striking symptom requiring interpretation, it is the collateral symptoms that clinch the diagnosis. We must not ignore the cramp-like spasmodic and persistent peri-umbilical pain of intestinal obstruction. If it occurs after abdominal section, even though the patient is still in the hospital, one should think of adhesive bands causing obstruction. Reoperation is less dangerous than a purgative. Who can differentiate the pain of mesenteric thrombosis from intestinal obstruction? The court of immediate appeal is exploration.

The whole subject of pain must be considered from the neurologic standpoint and is the most fascinating study. From the classical relentless pain of tic douloureux to the plebian painful heel, the immense network of sensory nerves ramifying to every area, associated with every function and distressed by every dysfunction weaves a tangled web. Only by knowledge of the nervous distribution can one correctly interpret the occult origin of pain. Witness the referred pain to the inner side of the knee along the branch of the obturator nerve in hip-joint disease in children.

After all it is the relative significance of pain that we must take into account. Disaster follows on our lack of interpretation of its importance. Excruciating pain in the shaft near the diaphysis of one of the long bones in a child in connection with chill and high fever imperatively calls for the recognition of acute osteomyelitis. It is murderously disguised as rheumatism, and, if not correctly interpreted and promptly treated by early evacuation, acute bone abscess occurs and long continued reparation for the bone destruction and sequestration of delay displays its weary length. The general symptoms are so alarming and overwhelming that no time should be lost in giving vent to the infection in order to prevent widespread death of the medulla. In such urgent circumstances the bone must be opened immediately even if you have to use a gimlet. This statement will be recognized as the plea of that greatest of American teachers of surgery, John B. Murphy.

Pains in the abdomen are not all due to visceral disease. Tuberculosis of the spine with pressure on one of the spinal nerves can give such pain over the distribution to the abdominal wall as to delude the unskilled observer. The abdomen has been opened for the unilateral referred pain from a carious spine that only required a well fitting brace.

One must think in afebrile abdominal cases of the gastric crises of tabes dorsalis, of plumbism, of the abdominal anginoid attacks in arteriosclerosis.

Pain is a monster that may be so insistent as to compel our greatest interest and yet it may be a gay deceiver. We have learned to be so suspicious of the pain complained of by the neurotic that occasionally their cry of "wolf" is unheeded. The usual pitfall is mortifyingly in the other direction. The constant burning pain over the right iliac fossa that has been diagnosed chronic appendicitis and operated upon elsewhere, often

comes to you with the selfsame pain unrelieved. By their scars ye shall know them. A case that has never had a bona fide acute attack should make us counsel the patient to get relief of the pain by other than surgical measures. We have it upon very highest authority that there is no closed season for the neurasthenic. They are as sheep in wolves clothing, and even deceive that good shepherd, the family physician.

Pain in these unfortunates may depend upon an abnormal personality rather than an organic abnormality. They need a physician who can "raze out the written trouble of the soul."

He is the best surgeon who is able not only unerringly to recognize the surgical significance of pain, but who also will clairvoyantly divine the significance of non-surgical pain.

PAIN ASSOCIATED WITH SURGICAL LESIONS OF THE EYE[1]

By JAMES M. PATTON, M.D., F.A.C.S., Omaha

ALTHOUGH the failure to recognize the significance of pain secondary to surgical lesions of the eye may only in very rare cases result in the death of the patient, there are numerous cases where the pain is so severe and far-removed from the involved eye and the general symptoms so prostrating that the vision of one or both eyes may be irrecoverably lost before either the surgeons or patient awakes to the fact that the eye is the source of the trouble.

This is especially true in acute inflammatory glaucoma. In the majority of cases the diagnosis is easy enough. The patient is well aware that the eye is exquisitely tender and that the severe neuralgic headache is directly associated with the ocular condition, but every oculist of experience has seen cases where the attack is ushered in by a most severe prostrating neuralgic sick headache. Nausea and vomiting may be a prominent symptom with or without severe epigastric pain. The patient is glad to lay perfectly quiet with the eyes closed, not even suspecting that they are the source of the trouble. Any case of otherwise unexplained neuralgic headache with or without gastric symptoms should suggest the possibility of an acute glaucoma.

Pain of a similar character though less severe associated with a blind eye, especially of long standing, may indicate the presence of an intra-ocular tumor, and if the oculist cannot definitely rule it out the general surgeon first suspecting the condition should lend his moral support on the side of enucleation. A sudden pain in or about the eye during the course of pneumonia, typhoid fever, etc., should at once suggest the possibility of a beginning metastatic panophthalmitis. Here again the symptoms may be masked and as the progress is often very rapid an early grave prognosis is indicated.

A beginning iritis or iridocyclitis may be mistaken for a simple conjunctivitis and much valuable time and even useful vision lost unless the surgeon remembers that the headache of which the patient complains, radiating over the side of the head and worse in the after part of the night, is most suggestive of iritis. If on palpation he finds a zone of tenderness just back of the cornea he can feel sure that the ciliary body is also involved. Pain on rotating the eyeballs usually means an inflammation of Tenon's capsule and indicates a prompt search for some focus of infection. A retrobulbar inflammation of the optic nerve may be painless, but a rapid decrease of sight associated with deep pain back of the eye should suggest this condition or possibly a deeply seated orbital tumor, although this condition can usually be recognized by the displacement of the orbital contents. The pain of an orbital abscess is more severe than either of the above but of the same character, usually associated with more or less general prostration. Corneal infections may cause pain out of proportion to the appearance of the lesion. Here again the pain is radiating in character, but there is usually sufficient local discomfort to call attention to the area involved.

[1] Presented in the symposium on pain before the Clinical Congress of American College of Surgeons, Chicago, October 22-26, 1923.

The headaches and eye aches resulting from errors of refraction and muscle unbalance are as a rule easily recognized from the history, although they may be so severe at times as to be quite incapacitating and demand prompt recognition and relief. Two forms of pain from this source may be misleading, viz., deep cramping aches at the back of the neck and between the shoulder blades, the result of faulty posture, and the severe often one-sided headaches associated with so-called scintillating scotoma, both calling for careful attention to refraction and muscles.

In conclusion, let us remember that a severe neuralgic headache with or without nausea should suggest the possibility of acute glaucoma; that the pain of iritis is worse in the second half of the night; that sudden pain in one eye during the course of a general septic process may mean a destructive panophthalmitis; that pain in or about an old blind eye is an intra-ocular sarcoma unless proved otherwise; and that even though the pains indicating the lesser lesions mentioned may not indicate such grave conditions, they are worthy of recognition by the general surgeon and his prompt intelligent advice will entitle him to the heartfelt gratitude of his patient.

INTRACRANIAL PAIN[1]

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INTRACRANIAL pain is common. It may be but a mild toxic headache or an unendurable pain of meningitis. It may be a severe intermittent pain due to brain tumor or the constant, bursting pain of an infectious exudate. It may be so mild that the individual pursues his task, or so severe that he seeks a darkened apartment, buries his head in the pillows and shrieks aloud from intolerable suffering. It may be but an inconvenience resulting from indiscretion in diet or it may be so severe as to clearly indicate impending death.

Pain anywhere probably is always caused by absorption of toxic products by sensory nerves, or by their traumatism. Usually both these causes are active at the same time. This pain may be due to pressure, to infection, or to pressure plus infection. Environment has much to do with the ease with which pressure pain is produced. In regions where loose tissues prevail, as in the neck, the pressure of a growing tumor, or of an exudate, pushes the sensitive nerves aside, and pain is delayed. Inside the cranium conditions are different. No structure within the skull may be pushed aside without causing definite traumatism and positive pain. The encephylon is covered by the meninges, the dura of which is almost wholly inelastic. It is further encased by the skull which provides absolute resistance to expansion. Increased intracranial contents will, therefore, mash and traumatize all structures against the hard and non-resistant inner table of the skull.

The dura mater, because of its position next the skull, is the part most easily traumatized by intracranial pressure. It is also the only intracranial tissue that is to any degree endowed with sensation. The brain itself is wholly without sensation. The pia mater has but few sensory nerves, while the arachnoid is wholly insensitive. In the dura mater, therefore, all intracranial pain originates. The actual cause of the pain often lies in the deeper structures of the brain, but the influence of that cause must in some way reach the dura mater before pain may be produced. Intracranial pain, it would seem, is always in the end an affection of the dura mater. The dura is the sensory organ of the brain.

What pathological conditions and diseases are foremost in the causation of such pain? The contents of the skull cavity are normally sterile. So long as this sterility is maintained, intracranial pain may not occur. The physiological status of the brain is disturbed chiefly by the entrance from without of infection or the products of infection. The healthy brain is menaced by this invasion from two sources,

[1] Presented in symposium on pain before the Clinical Congress of American College of Surgeons, Chicago, October 22-26, 1923.

namely, through the blood stream, and by direct entrance through the skull itself. The blood stream carries disease, or the products of disease, from distant foci of infection. Direct invasion of the intracranial contents may result from injuries, but more often from necrosis of bone due to suppuration in one or more of the numerous sinuses or cells which lie between the tables of the skull, and which are in direct communication with the nose and ears, which, frequently, are mere containers of sepsis.

Intracranial pain, therefore, is due usually to a definite assignable cause, and may often be traced to some external, clearly understandable source. Its extrinsic origin is certain and usually definable. The paranasal sinuses and the mastoid cellular labyrinth are the commonest foci of origin. From these paranasal sinuses and mastoid cells, which in many individuals are so numerous and large as to surround great areas of the brain, infection products, often present there, rot their way through the thin inner table of the skull, and directly invade its sterile contents. Or, the intercommunicating blood stream may carry the infection from the septic cell to the sterile brain. The result is the same in either case. Inflammation is set up, inflammatory products are formed, intracranial pressure is increased, the sensitive dura mater is crushed against the skull, and intolerable pain is produced.

1. *Tic douloureux.* One disease, tic douloureux, which probably causes intracranial pain, although such pain is commonly recorded externally, is an affection of the gasserian ganglion, and has no definite pathology. Possibly tic douloureux should not be classified as a disease giving rise to intracranial pain. It certainly has not been proven that tic is the result of infection. It is, however, becoming increasingly clear that the real pathology, whatever it may be, is oftenest in the ganglion, and that the external pain of tic is but a reflex. Clinical experience, in dealing surgically with the disease, leads to the conclusion that both the disease and its terrifying pain are in reality inside the skull.

2. *The circulation in the brain of toxic substance in the blood.* A long list of acute and chronic general diseases are responsible for this type of intracranial pain. Common headaches, often severe and recurrent, reducing health to the point of invalidism, are due to this cause. Toxins arising from indigestion or constipation circulate in the blood stream of the brain and poison, temporarily, the sensitive dural nerves. No doubt transitory exudates occur simultaneously, and thus both toxicity and pressure act together as a cause of pain. Migraine, worst of periodic headaches, should probably be placed in this class of ephemeral, toxic headaches. Meningitis, typhoid fever, measles, small pox, and erysipelas head the list of acute diseases. All these may be heralded and accompanied by intolerable headache. The toxin is absorbed by the sensory dural nerves, and the increased blood supply plus the accompanying exudates traumatize the dura by crushing it against its osseous capsule. Among the chronic diseases of this class Bright's disease and glycosuria are best examples. The circulation of syphilitic poisons in the brain may cause headache in the earlier stages, but later exudates and tumors of luetic origin are chief factors in the pain production, and the same may be said of early and late tuberculosis.

3. *Tumors of whatever kind and cause, as gummatous, tubercular, gliomatous, cancerous, etc.* The severity of the pain may be out of proportion to the size of the tumor. Its location may have much to do with the amount of suffering. A small tumor which obstructs the interventricular circulation may produce more pain than a tumor many times its size but placed differently. The amount of brain displacement and consequently the degree of intracranial pressure is more the cause of the pain than is the nature of the tumor itself. Thus the fact that a gumma is large is more reason for pain production than that it is of syphilitic origin. For the same reason a brain abscess causes pain, not so much because it is an abscess as that because of its size it crushes and traumatizes the sensitive dura mater. Other symptoms may be present which will differentiate a brain tumor from a brain abscess, but pain of similar character and severity is apt to be the same in both. Intracranial pain is a symptom of first importance in both brain tumor and brain abscess. Pain,

choked disk, and vomiting are the most constant symptoms of brain tumor and brain abscess, and of these the one most often present is pain, severe, torturing, and more or less constant.

Intracranial pain is undoubtedly less frequent today than formerly. It has to no inconsiderable extent been prevented or conquered by modern medical and surgical science. The internist and laboratory expert, working together, have discovered the cause and nature of blood stream infections acting on the brain to cause pain, and have in several pain producing diseases, notably in syphilis and diabetes, robbed these diseases of their most terrorizing symptoms. Surgery also has added greatly to the relief of intracranial pain when due to tumor growths or fluid collections. The splendid technique of Fellows of the Royal College of Surgeons of England, and the equally brilliant work which is being advanced by Fellows of the American College who are specializing in brain surgery bids fair to eliminate in the near future a yet greater amount of intracranial suffering by means of surgical measures.

PAIN IN THE EAR[1]

By FRANK M. SULZMAN, M.A., M.D., F.A.C.S., Troy, New York

WHEN a patient comes to us complaining of pain in the ear, it is important to know whether the pain is due to a lesion about the ear or is a reflex pain due to a lesion in some other part of the body causing this phenomenon. Much suffering is prevented if care is used in determining the cause of the pain, for operative measures about the ear in a patient suffering from reflex pain would only make the condition worse, and until the original focus of trouble is properly taken care of no permanent relief can be expected. As we all know, pain is one of the most common symptoms of pathological conditions arising directly in the external, middle, or internal portions of the ear. From the infective process in any of these portions of the ear come the more grave complications that arise in ear work, to relieve which requires at times very extensive surgery of the mastoid sinus and intracranial structures.

To diagnose early and locate the cause of pain in the ear is of prime importance, as nearly all pain about the ear proper is from pressure; relieve this pressure, institute adequate drainage, and fortunately the majority of the acute ear cases complaining of pain promptly recover. Not to do this causes untold discomfort and adds a dangerous factor by causing extension of the trouble to structures adjacent to the ear, which, when involved, increase enormously the danger to life and prolong the period of suffering with its extra expense and loss both from the business and from other standpoints. It is of interest to note the different names patients and otologists use in describing the pain associated with ear lesions. For example: we have pain described as "dull," "aching," "spontaneous," "boring," "lance-like," "throbbing," "darting," etc., each meaning to either patient or doctor much as regards the type of the condition likely to be met.

In contradistinction to pain in other parts of the human body, it has long been known to the laity that aside from the intense pain they suffer, earache is associated with danger, and for this reason we fortunately see our ear cases in early stages.

Several factors enter into the degree of pain either experienced or found on examination: first, the patient; second, the type of infection; and third, the structure of bone, whether soft or pneumatic or the ebony structure seen not uncommonly.

We all know a certain type of patient greatly exaggerates any symptom, while the opposite type seen not infrequently, make little of any illness, and we marvel at times how the latter have stood the pain when examination reveals the advanced condition. I was once hurriedly called to the hospital to see a man, whom, his family doctor told me, he had seen the night before and realizing the serious con-

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dition present insisted that he go to a hospital. He did not consent until the next morning and when I saw him in the emergency room he was semiconscious, both ears discharging while pressure over either mastoid would rouse him for a few minutes. He was operated upon for a double mastoid. After making the incision and starting to use my periosteal elevator, I noted that the bone would move under my instrument. I stopped and raised the flap with dissection and with my tissue forceps took out a sequestrum that represented a good portion of the temporal bone. The mastoid on the opposite side showed extensive necrosis but not the same picture. He was not expected to recover, but he did so and when asked why he deferred seeking relief, told me he did not mind the pain.

Another case seen in a comatose state died while I was endeavoring to stimulate and improve her condition which did not justify attempting a mastoid operation. Death occurred within an hour after entering the hospital. When the mother was asked why she allowed the daughter to progress to such an extreme condition, she replied that the fear of the hospital was the only reason; yet the pain and suffering must have been intensely severe. Further inquiry as to any basis for such fear of a hospital revealed nothing. The antipode of this extreme is the other type, the one who complains of intense pain when a speculum is put in the ear for routine examination, no inflammatory condition being present. A marked difference is here, you will all agree with me. Yet both types are found, I know, many times by those doing ear work. In estimating the value to be placed on the degree of pain, consider your patient; it will be of untold value to you in many instances. We often when examining the ear, should place value on the facial expression showing real distress rather than fear. The type of infection seems to change the amount of pain to a marked degree. In cases with marked destruction of tissue from a streptococcus capsulatus infection the pain may be mild or altogether absent; the same holds true with many of the tuberculous type. The third element entering into cause of pain, structure of bone, will be spoken of later.

Pain can aid us in our diagnosis and when properly interpreted holds a prominent place in our regular routine in all ear work. If in the examination the patient endeavors to move away when the auricle is touched, if pain be constant or worse when the mouth is opened or closed the lesion is in the external canal and we shall be safe in making a diagnosis of acute otitis externa. Cases of acute otitis externa that are not seen early may cause a good deal of swelling over the mastoid area with the usual signs of mastoiditis, and we should carefully examine the canal and drum membrane in all.

If no pain is noticed when the finger is pressed over the mastoid, but pain is present upon pressure up and inward under the lobe of the ear, the pain worse at night and in the recumbent position, the trouble is in the middle ear and will be verified by further examination of tympanic membrane.

Mastoid pains vary with the character of bone which is being dealt with. The pneumatic and ebony type will each have special symptoms. In the soft pneumatic bone pain on pressure will be great, but the patient will complain very little, as pus has found an easy passage toward the cortex. In the ebony type the patient complains of much pain and pressure will not reveal it even until later in the course of the disease. No matter what the history or symptoms, as a regular procedure in all inflammations about the ear, test out carefully for any of the painful points with which you are all familiar in the mastoid region, especially note the area over antrum and tip. Do not consider your examination made unless you have considered these two as a regular duty. There is still another type of pain about the mastoid, extremely troublesome when found, painful on pressure. The patient complains of severe distress as in an ordinary mastoid infection; yet examination of the canal and middle ear shows no true ear involvement. We have here an otalgia to deal with and the foci of infection must be looked for in the tonsils, teeth, or other parts of the body. A mastoid operation is unnecessary. With a history of pain followed with or without discharge from the ear, with a marked rise of temperature, 104–105 degrees,

dropping to nearly normal in a short time, this to be repeated in 24 hours, we must consider a possible sinus thrombosis.

A case with headache and vomiting and a history of previous ear discharge with or without pain directly in the ear, should warn us of a likely intracranial complication, meningitis especially. Pain is one of the early symptoms of brain abscess and we should remember that the site of pain is no guide to the location of the abscess, occipital pain being present in many cases of temporosphenoidal abscess while frontal pain is found in cerebellar involvement. Aphasia if present would aid us in locating the abscess in the left side. In all these infections associated with deafness, vertigo, nausea, and nystagmus, examine the labyrinth for cause of your trouble.

PAIN IN THE JOINTS AND BACK[1]

By R. D. KENNEDY, M.D., F.A.C.S., Globe, Arizona

WHEN I was asked to speak on the subject of pain in the back and joints, I came to the conclusion that the committee must have been studying Arizona's compensation laws. Because of our inadequate compensation laws, I know of no other place where pain, especially in the back, can be and often is so highly capitalized. As pain is a subjective symptom and is caused by such a variety of lesions, in order to properly interpret it, we must avail ourselves of all the information we can acquire leading up to its cause. This information is gained through a careful and painstaking history, noting particularly previous illness, mode of onset of the present attack, duration, and the noticeable symptoms.

Next will come the physical examination. In all cases in which pain in the back is the chief complaint, the patient should be stripped, and his general condition noted—any deviation from normal in the contour of the spine, tenderness over the spinous processes, and any evidence of muscular spasm. The patient should be made to put the spine through the full range of motion, and any limitation or increase of pain on any of the movements noted. Proper X-ray pictures should be taken, and any other laboratory facility employed which will help us to a better understanding of our case.

Cases with pain in the back may be divided as to cause into several groups: those due to injury; those due to arthritis; those due to traumatic neurosis; those due to new-growths; those due to uterine, pelvic, or abdominal disorders; those due to postural or mechanical strain; and those due to a myositis of the muscles of the back.

Traumatic backache may be divided into cases of fractures and sprains. It is as a rule, no trouble to diagnose serious fractures, but it is possible to diagnose many minor fractures only by the X-ray, as fractures of the laminæ, spinous or transverse processes, as a rule, cause no deformity, and are frequently overlooked. All these cases should be X-rayed in the lateral and anteroposterior direction.

I have seen cases where one or more of the lateral processes of the lumbar vertebræ were broken in which the patient lost no time from his work, while others with apparently a much less severe injury of the same character would be away from work for months, or until a settlement was made.

Simple sprains of the spine are common and the symptoms vary according to the degree of injury or the site. Usually a history of a blow, torsion, or sudden and unusual muscular stress can be elicited. They are most common in the cervical and lumbar regions. The onset is sudden, and the pain is increased by certain movements which put the injured parts on a stretch. In the lumbar region, the pain may radiate downward into the thighs and buttocks, and even into the calves of the legs. The X-ray is negative.

Where compensation is involved, this kind of case is often difficult to treat as many of them refuse to get well until settlement is made. The pain due to adhesions is usually

[1] Presented in the symposium on pain before the Clinical Congress of American College of Surgeons, Chicago, October 23–26, 1923.

located in the region of the seventh cervical or dorsolumbar junction, and over the fifth lumbar vertebra and sacrum. In the absence of arthritis or other demonstrable cause, this should always be borne in mind.

Arthritis of the spine occurs either primarily in the spine or in connection with a general arthritis. The onset may be insidious or abrupt. There is a gradually increasing stiffness of the spine with pain radiating forward along the ribs and downward into the buttock. When the involvement is in the lumbar region, the lumbar curve is flattened, the muscles are held tense and movements of this portion of the spine are painful. When the dorsal region is involved, rotation becomes restricted and chest expansion diminished. The pain is increased by jars, and there is a gradually increasing kyphosis.

Altitudinal strains are a cause of a high percentage of backaches and are due to a faulty deflection of body weight causing constant strain to spinal joints, ligaments, and muscles. The causes are most often the shortening of a leg, flat foot, shortened posterior muscles in the leg, or a very large abdomen.

The deviation may be either lateral or anteroposterior. If it is in the lateral direction, the pain is usually unilateral and is most often in the lower half of the spine. It is more frequent in women than in men. It is aggravated by standing and relieved by sitting. The pain is most often on the convex side of the curve. In cases of some standing, there is a shortening of the tissues on the concave side and a consequent limitation of motion in the opposite direction. If the deflection is in the anteroposterior direction, the pain is, as a rule, associated with standing, walking, sitting or lying down. It may be located in any part of the spine, but is most often in the lower half. The pain is a dull, dragging one. The patient is relieved by having the physiological lumbar curve supported. The pain may be unilateral or bilateral and is frequently located over the region of the sacro-iliac joint where tenderness may be elicited. These are often diagnosed as sacro-iliac strains. The X-ray shows no displacement.

Abnormally long transverse processes of the fifth lumbar vertebra are often a cause of pain in the back. When the extremity of such an elongated process comes in contact with the base of the sacrum during lateral flexion, an irritation is set up causing a localized outgrowth of bone to develop from the sacrum with the formation of a bursa or false joint. If the condition is unilateral the pain is relieved by bending in the opposite direction.

In some cases the transverse process of the fifth lumbar vertebra fuses not only with the sacrum but also with the ilium. This condition, combined with an osteo-arthritis of the sacrolumbar joint may result in the reduction of the size of the intervertebral foramen which transmits the anterior diversion of the fifth lumbar nerve, producing pressure on the nerve, and pain. The fourth sacral nerve which descends in front of the lumbosacral transverse articulation may also be pressed on, causing pain along the course. The X-ray will show many cases of sacrilization of one or both processes causing no symptoms.

Following railroad accidents, being buried by a fall of dirt or other similar accidents, pain in the back is often complained of without demonstrable lesions in either the spine or cord. The pain is usually not well localized and is often of a radiating character. It may be accompanied by partial or complete rigidity of the spine. There may be areas of anæsthesia or hyperæsthesia. The pain often increases some time after the accident, instead of improving as expected and is associated with great depression. Patients become irritable, sleepless, and are unable to concentrate. In some cases there may be functional paresis with loss of control of the bladder and rectum.

PAIN ASSOCIATED WITH GYNECOLOGICAL AFFECTIONS[1]

By RICHARD R. SMITH, M.D., F.A.C.S., Grand Rapids, Michigan

IN dealing with so large a subject in so brief a space of time many general statements are necessary and such are apt to be unsafe, since the dissentor may readily find instances to disprove them. I trust you will bear this in mind in reading this article. Certain general conceptions as to pain in gynecological affections are valuable, however, in giving us a lead, though they seldom establish the final diagnosis.

Pain arising from lesions in the pelvis is located in the lower abdomen, the groins, the external genitalia, the uterus, and vagina, and less often in the rectum and sacrum. There is very little tendency to radiate, as compared for example with that arising from obstructed biliary or urinary passages. When it does it extends down the thighs or upward toward the diaphragm and loins. It seldom leaves any doubt as to its origin. Such radiation, when it does occur, may sometimes be taken as a sign of severity or of unusual sensitiveness.

Gynecological affections seldom give rise to backache. When they do, the pain is more apt to be in the sacrum than in the lumbar region. The cause of the common lumbar backache of women is to be sought in fatigue, in faulty posture, and occasionally in arthritis, and not in the pelvis. Headaches of all descriptions, and pain in remote parts of the body are not caused by gynecological affections. Such remote pains and backache are frequently associated with pelvic difficulties, but not caused by them, and it is imprudent to promise their relief by surgical or other gynecological measures. The old time so called "reflex pains" are a matter of historical interest only.

Simple ovarian tumors do not cause pain, but there may be discomfort when they reach a large size. Pain occurs only when there are complications, such as twisting of the pedicle or infection and inflammation about the tumor involving the peritoneum. Likewise fibroid tumors do not, as a rule, cause pain until they reach a large size when they may do so for mechanical reasons. Its cause, when present, is commonly found in some complication, such as infection and necrosis in the degenerative process of such tumors. We are dealing here, however, with a muscular organ subject to painful contractions in the effort to expel. Uterine tumors, especially intrauterine ones, may thus cause intermittent pain. There is a type of fibroid uterus—a small uterus with many fibroids—which is tender and painful, the cause being not readily apparent.

Malignant tumors of the ovary seldom cause pain or discomfort until they are far advanced, and until there is a large amount of ascites. Cancer of the cervix and uterine body in its early stages is unfortunately not painful. Its presence is a sign of advanced disease.

Simple retroversions cause no pain, and for that matter, no other disturbance. Extreme flexion occasionally causes discomfort. It is often present when there is subinvolution or other uterine pathology increasing the size of the body. An incarcerated uterus usually gives rise to much pain. A uterus fixed in retroversion by adhesions, with the attendant disease of the appendages, is often painful.

As to prolapse, even a moderate degree may cause discomfort when the patient is up and about, and an extreme degree almost always does so sooner or later. The attendant cystocele and rectocele, especially the former, add materially to this discomfort.

Acute salpingitis causes moderately severe pain compared with other acute abdominal infections. In infections going out from the uterus or tubes, pain means an involvement of the peritoneum though the peritonitis may be well circumscribed.

The acute pain of a ruptured ectopic pregnancy is one of the severest with which the gynecologist has to deal. It is at first usually general in character, later located in the lower abdomen and in one or the other groin. The tube from which the hæmorrhage has occurred may thus be safely surmised, though there is an occasional exception. Pain in the rectum

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from the filling of the cul-de-sac with blood is a very common symptom and may be used to strengthen a diagnosis, since its occurrence in other pelvic conditions is not so frequent.

Simple lacerations of the cervix do not cause pain. When attended by induration and inflammation there is sometimes an indefinite discomfort in the pelvis. It is rather remarkable that a badly lacerated, indurated, discharging cervix causes so little discomfort.

Old lacerations of the perineum cause no pain, but an accompanying rectocele may give rise to discomfort. Most of the disturbance from the lacerations of childbirth come from a consequent cystocele. A urethrocele or eversion of the sensitive mucosa of the urethra—conditions which are often overlooked when an examination is made with the patient relaxed and in a prone position—sometimes gives rise to marked soreness and discomfort.

Dysmenorrhœa may occur in patients with a uterus apparently sound, as well as in patients with the so-called underdeveloped uterus. Its direct cause is spasm of the uterine musculature, and the disturbance is a functional rather than an organic one.

Painful affections of the vulva need no mention in so brief a paper.

We have mentioned pain in its relation to those lesions of the female genitalia the correction of which is the peculiar function of the gynecologist. Often, however, we find pain, when so far as we are able to determine, the organs are normal or present lesions that should not be painful. The pain, so far as the location goes, simulates very closely that attending real lesions of painful nature. There is nothing that requires greater diagnostic skill on the part of the gynecologist than the accurate estimating of the physical condition of the pelvic organs and the part that the nervous system is taking in the symptomatology.

The term neurosis is almost as general as the term disease, so varied are its manifestations. We must bear in mind first that we are dealing with sensitive functionating organs and not inert ones. Our conception of what constitutes a neurosis may vary greatly; gynecologists are not neurologists nor are most of them profound in their knowledge of neurological literature, but in a general way it may be said that two principal factors enter into the production of pain in the nervous neurotic woman. The first is an over-sensitiveness of the patient, so that organs ordinarily functionating painlessly now do so painfully. The second factor lies in the mind of the individual, whose faulty conceptions as to the integrity of her sex organs and the attending anxiety in regard to them exaggerate normal sensations into those of pain or discomfort. There are almost always other manifestations of the neurotic condition. We are dealing then with a functional disturbance of the nervous system, of which pain in the pelvic region is but a symptom. The success of the gynecologist in his particular field of endeavor will depend largely upon a rational understanding of such situations and his determination to refrain from instituting surgical measures.

PAIN DUE TO PATHOLOGICAL CONDITIONS OF THE GENITO-URINARY TRACT[1]

By A. J. CROWELL, M.D., F.A.C.S., Charlotte, North Carolina

KIDNEY pain is due to some stimulation of the sensory nerve supply of the kidney. Severe kidney pain is the result of some acute process and is divided into the inflammatory and non-inflammatory types. The inflammatory type is constant, aching in character, increased by palpation and percussion while the non-inflammatory type is more sudden in onset, more severe and paroxysmal in character and disappears more suddenly than that due to inflammation. The patient lies on the opposite side to prevent pain in inflammatory conditions and on the affected side when pain is due to kinked ureter in nephroptosis, or any non-inflammatory condition causing sudden ureteral occlusion. Inflammatory kidney pain is increased more by deep breathing than by motion. That caused by kidney congestion is better in the morning and worse in the evening. The latter is not eased by fixation and is sensitive to deep pressure.

Pain in the kidney region associated with local tenderness signifies kidney involvement. Referred pain in kidney lesions is felt in the lower iliac and suprapubic regions and to the outer, middle, or inferior thigh. Pain in the penis, scrotum, perineum, inner aspect of the thigh, or lower sartorius indicates lower ureteral involvement. If felt in the latter areas in renal colic with increased frequency of urination, without pain during the act, the stone is nearing the bladder. In high ureteral involvement the skin of the scrotum is not painful to pressure but the deep tissues are.

Probably 50 per cent of right-sided pain is due to kidney lesions. In movable kidney, pain is increased by standing and relieved by lying down. Torsion of the renal vessels in Dietl's crises increases intracapsular pressure and causes kidney soreness for some time following the attack.

Renal infarction pain is sudden in onset, burning in character, free from paroxysms, and does not radiate into the inguinal region or the genitalia. It is increased by motion and relieved by reclining on the affected side.

The pain in perinephritis is severe and located in the lumbar region. If the lumbar plexus of nerves is involved, the pain is referred to the knee and lower thigh, and especially so, if the abscess is located at the lower pole of the kidney. If the abscess is at the upper pole, the intercostal nerves are involved and the pain is referred to the area of their distribution. If the pain and tenderness are sharply limited and associated with œdema, the infection is perinephritic. In suppurating conditions of the kidney, pressure in front causes considerable pain while in perinephritic abscess the tenderness is greater in the back.

Severe pain in the kidney region following trauma when accompanied by hæmaturia means rupture of the kidney. If paroxysmal in type, it signifies that the rupture has extended into the kidney pelvis.

Pain is present in the terminal stages of tuberculosis of the kidney. It is increased by pressure on the anterior wall, the paraumbilical, subcostal, and lumbar regions. It is associated with frequency of urination and occurs before and after voiding. In far advanced cases, ureteral colic is produced by the passage of blood or pieces of necrotic tissue.

Pain is present in about 75 per cent of the cases of pyelitis. It radiates from back to thigh, perineum, and genitalia; or upward to the epigastrium and shoulder. Upper ureter involvement causes pain similar to that of diseased kidney. Pain in the kidney zone prior to its presence in the ureteral zone means kidney lesion. Kidney zone pain persists after the ureteral obstruction has been removed, while the ureteral zone pain disappears spontaneously.

A renal calculus without infection does not produce pain unless blocking of the ureter occurs. The pain is due to increased intra-

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capsular tension. If the capsule is thickened and non-elastic when subjected to pressure the pain is severe. It is constant when accompanied with pelvic infection and paroxysmal when the stone is too large to enter the ureter and is so located as to have a ball-valve action. When occlusion occurs, the pain is sudden in onset and relief; is unilateral and radiates to crest of ilium, anterior abdominal wall, groin, and testicle on the same side; and occasionally extends down leg to toe. It is relieved by pressure and aggravated by motion.

In ureteral calculus, the pain is present in both the kidney and ureteral zones. Distention of the ureter itself has something to do with the production of pain. Changing position of pain as the stone moves down the ureter is clinical evidence of this fact. Pain due to complete ureteral obstruction gradually becomes less marked and disappears unless complicated by kidney infection. This is due to the progressive decrease and final inhibition of the urinary secretion.

Spasm of the ureter will produce an acute hydronephrosis and paroxysmal pain. The same kind of pain will result from occlusion of the ureter with calculus, blood clot, detritis, stenoses of the ureter or ureteritis. When the ureter is inflamed, pain is elicited at the brim of the pelvis by deep palpation, and by rectal or vaginal examination.

Bladder lesions produce both frequent and painful urination when the lesions are located in the trigone; otherwise, they are practically painless. The trigone is the only part of the bladder which is extremely sensitive. A vesical calculus will produce agonizing pain at the end of urination, especially if associated with trigonitis. If the bladder is adherent to the rectum or sigmoid, bowel action will produce urinary tenesmus and bladder pain. Adhesions to the uterus and tubes will produce sever pain during menstruation, pregnancy, and sexual intercourse.

Bladder pain is of two types: constant and paroxysmal. The constant type is felt behind the symphysis and indicates a severe inflammation which extends into the muscular walls of the bladder. Paroxysmal pain occurs just before and at the end of urination. Bladder lesions causing pain are vesicourethral fissures, cystitis, pericystitis, tuberculosis, tumors, calculus, distention, and rupture of the bladder. Pain due to ruptured bladder is severe and is located in the lower abdomen. It is sudden in onset, and follows trauma. The desire to urinate is constant but not relieved by attempts to void.

Tumors of the bladder cause pain by obstructing the ureter and distending the renal pelvis or by blocking the urethra and producing retention of urine.

PAIN IN THE UPPER ABDOMEN AND CHEST[1]

By CHARLES H. PECK, M.D., F.A.C.S., New York City

PAIN in the upper abdomen or chest as the initial or outstanding symptom is common to many pathological conditions not always easy to differentiate.

The first consideration should be to determine whether the condition is medical and amenable to proper medical treatment, or whether the pain indicates the onset of some serious surgical condition which may demand prompt operative relief. Until the latter possibility is safely excluded, the administration of morphine or sedatives which may mask important symptoms and obscure the diagnosis, should be avoided.

A number of non-surgical conditions may cause upper abdominal pain; as, simple gastric indigestion with which most of us have had personal experience, food or ptomaine poisoning, or the ingestion of poisonous drugs or substances.

Pain referred to the upper abdomen may be caused by: (1) Conditions within the chest or about the diaphragm, as pleurisy, pneumonia, pulmonary infarct or abscess, thoracic aneurism, mediastinal inflammation or neoplasms, angina pectoris, and the pain of venous congestion of the liver and abdominal veins in cardiac decompensation, the pain of a perihepatitis, which I have seen caused by the rough nodules of a liver carcinoma projecting on the convex surface against the diaphragm. I was called to a distant city in consultation on such a case, the cause of the obscure, persistent pain having been unrecognized. (2) Pylorospasm and the condition known as writhing duodenum. I removed the gall-bladder in a case of the latter malady and later another surgeon performed duodenojejunostomy, but the duodenum still writhes, according to the radiologist and the patient, a well known physician and old personal friend, who had had medical and diagnostic advice from many expert sources. (3) The gastric crises of tabes, for which I have seen gastro-enterostomy performed. (4) Peripheral nerve pains due to spinal nerve root pressure, to local neuralgia, or neuritis, to the early stages of herpes zoster.

Turning to surgical causes of upper abdominal pain, operative, or non-operative, lesions of the biliary tract deserve first consideration. Four general types of pain cover most of the cases:

Type 1. Typical biliary colic; severe, intermittent; with complete freedom from pain in the intervals; irregular in time of occurrence; with occasional slight jaundice after the attack.

Type 2. Acute gall-bladder distention with occlusion of the cystic duct; constant, severe, increasing pain and local tenderness, often with fever and leucocytosis. Some cases go on to acute cholecystitis, abscess formation, rupture of the gall-bladder or gangrene.

Type 3. Chronic variable pain with gaseous indigestion without jaundice or fever which may last for years before the gall bladder is suspected. We meet with many such cases.

Type 4. The classical picture of stone in the common bile duct, with intermittent pain, fever, chills, and jaundice.

Gall-bladder pain is generally to the right of the mid line at the costal margin. It radiates to the back, right shoulder blade and shoulder, or across the abdomen to the left. It may be maximal in the mid line, or quite atypical in location. I have recently removed a gall bladder full of calculi in a woman who, for 3 years had had acute attacks of upper abdominal pain always referred to the *left* costal margin with no local tenderness. Several excellent diagnosticians had considered the pain of neurotic origin.

Pancreatitis should be considered in close relation to biliary tract disease, with which it is usually associated. Sometimes the extension of pain and tenderness to the left or the greater severity of the symptoms make a preoperative diagnosis possible. More often the clinical picture is merged with that of the gall bladder. In the acute hæmorrhagic or gangrenous type, the violence of onset and course

[1] Presented in symposium on pain before the Clinical Congress of the American College of Surgeons, Chicago, October, 22–26, 1923.

may simulate acute perforated ulcer or mesenteric thrombosis. The disease may be of any degree of severity ranging down to moderate swelling discovered in the course of upper abdominal operations. Pancreatic calculi are rare.

Many cases of upper abdominal pain have their source at a distance. Appendicitis, acute and chronic, is the most frequent offender. Epigastric pain at the onset is so common as to be the rule in many acute cases. Pain in chronic cases is often referred to the stomach. Appendicitis is a frequent cause of chronic digestive disturbances which closely simulate those of ulcer, and of so-called pylorospasm. High retrocolic appendices, when diseased, cause pain closely simulating that of gall-bladder disease, or of right kidney lesions. I have removed an acute perforated appendix where for 7 years previously repeated attacks of pain had been persistently located high up to the left near the splenic region. The long appendix ran upward to the left, its tip across the mid line pointing toward the spleen. A number of expert diagnosticians on both sides of the Atlantic had repeatedly failed to recognize the cause of the pain, attributing it to a neurosis. The attacks never recurred after removal of the appendix. Pain of appendix origin can simulate almost every known abdominal lesion and must be included in nearly every table of differential diagnosis.

Gastric and duodenal ulcer with their classical syndromes and also their many atypical manifestations form a group about which pages could be written. The scope of this paper will not permit of a careful differential study of pain types and their pathological basis. It must suffice to say that peptic ulcer, whether of the chronic indurated type, the subacute, acute, or chronic perforating types, is one of the most frequent and important causes of upper abdominal pain. Gastrojejunal or marginal ulcer following gastroenterostomy causes severe and persistent upper abdominal pain. The pain of acute perforating ulcer is often intense agony associated with shock.

Pain due to lesions of either kidney is usually located in the lumbar region and radiates downward to bladder, genitals, or thigh. It may, however, in acute infections, especially of the unilateral hæmatogenous type, cause pain referred to the upper abdomen. If this fact is borne in mind and other clinical symptoms and signs are correlated, diagnostic errors should rarely occur.

Subphrenic abscess, liver abscess, and perinephritic abscess, must all be included among the conditions to be considered. I have observed and reported a case of acute hepatitis, with swelling of the liver, recent fibrinous exudate, and a hæmorrhagic serous exudate, explored as acute appendicitis with recovery and subsidence of the condition, which was probably an acute infection entering through the portal system. Thrombosis or phlebitis of the portal vein as a cause of upper abdominal pain, I have met with once at operation performed for supposed subacute perforation of a duodenal ulcer. The portal vein and its two hepatic subdivisions were hard cords; there was ecchymosis and some exudate in the adjacent retroperitoneum, but no general ascites. The patient, a physician and personal friend, is perfectly well 8 years after operation.

I have recently seen a case diagnosed as septic thrombus of the portal vein, but unverified by operation or autopsy, go from acute onset to fatal termination in 3 days, the temperature repeatedly above 106 and once reaching 108.8 degrees.

Thrombosis of the mesenteric vessels, one of the acute abdominal tragedies, is often heralded by upper abdominal pain. I have recently explored a case within 8 hours of onset, with gangrene of the entire length of small intestine. Thrombosis of mesenteric branches with localized gangrene may occasionally be amenable to surgical relief.

Enlargement of the retroperitoneal glands due to tuberculosis, Hodgkin's disease, sarcoma, or metastatic growths, may cause upper abdominal pain, though it more often centers lower down. Malignant disease of the stomach, liver, gall bladder or ducts, pancreas, or colon, is a common cause of upper abdominal pain. Peritoneal adhesions in the region of gall bladder, duodenum, or stomach, post-inflammatory or postoperative, are a fruitful source of upper abdominal pain of variable

type and severity. To attempt a study of the symptoms and diagnosis of these conditions is quite beyond the scope of this paper.

Small epigastric herniæ in the mid line above the navel, are not uncommon causes of pain, and the rarer subdiaphragmatic herniæ and internal herniæ in the Fossa of Treitz, must occasionally be reckoned with.

Aneurism of the abdominal aorta or its branches may cause severe pain from pressure or erosion of vertebræ, or acute pain from rupture. Osteomyelitis or neoplasm of vertebræ or ribs, including metastic disease, must also be remembered.

I have discussed only the conditions which I have had the opportunity to observe personally and have undoubtedly omitted many important causes of upper abdominal pain, emphasizing, naturally perhaps, surgical more than medical etiology.

One might well think that 30 years' experience in active hospital practice in acute surgery should make the reading of upper abdominal painful conditions an open book, but I must confess that it is seldom that a week does not bring its puzzles in diagnosis and errors in interpretation. Upper abdominal pain must never be taken lightly or casually. Many cases are due to lesions which call for prompt surgical intervention. Many, when first seen by the surgeon, are masked by the previous administration of narcotics.

On the other hand surgical intervention without proper pre-operative study and diagnosis on the plea of exploration, may mean unnecessary operation for some trifling non-surgical condition and should not be tolerated.

In the last analysis, common sense and a broad knowledge of both medical and surgical conditions which may cause upper abdominal pain will protect both patient and surgeon against such errors.

DIABETES INSIPIDUS WITH ACUTE RETENTION IN PREGNANCY

With Report of a Case

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WHILE diabetes insipidus is not a rare condition, and is not infrequently seen in the larger clinics, the details of its etiology, possible complications, and treatment have not been thoroughly established. Each individual case should, therefore, be carefully studied. The combination of this, with the irritable bladder frequently seen with the enlarging uterus of pregnancy is something which seems not to be mentioned in the literature, and makes the following case, with its resultant difficulties unusual enough to warrant reporting.

Historically, symptomatic polyuria had been observed in ancient and medieval times; and in 1670 Willis found that some of these urines did not contain sugar. Near the end of the eighteenth century, Cullen and P. Frank (1) definitely separated them into two classes, with and without sugar. Since then, observations have been accumulating, and diabetes insipidus has been established as a clinical entity.

Clinically, diabetes insipidus is the disease or syndrome characterized by the passage of a large amount (5 to 10 liters daily) of dilute urine (specific gravity 1,001 to 1,009) having no albumin or sugar. Total solids eliminated are normal. There is, of course, a correspondingly large intake. The primary cause does not lie in the kidneys, which at autopsy are found normal. But the mortality is low and autopsies are infrequent. Theoretically, this polyuria is differentiated from a primary polydipsia with difficulty, but practically the distinction is usually clear. A chronic nephritis, interstitial or vascular in type, frequently gives a large output, but there is evidence of renal impairment, with lowered functional tests and elevation in the blood nitrogen. With true diabetes insipidus, there is (1) a possibility of concentration on a low intake, that is, the specific gravity is not absolutely fixed; (2) a possibility of concentration with pituitrin or with fever; (3) an excessive polyuria after the ingestion of chlorides, and (4) an absence of the theobromine effect.

Davis (1) found in 242 reported cases, that 75 per cent were in middle life, between the ages of 5 and 40, and that there were twice as many males as females.

Weil (2) described a family of 219 members, 35 of whom had diabetes insipidus.

Herrick (3), in 1912, reported his much quoted case, where the polyuria was stopped following a lumbar puncture with the removal of 5 cubic centimeters of fluid, with a severe reaction.

Cushing and others (4, 5), in 1913, brought forward the influence of the hypophysis in this condition and suggested that the hypophyseal hormone ascended by the tuber cinerum to act on the proximal mesencephalic centers themselves.

Since that time, many more cases of diabetes insipidus have been observed to be associated with changes in nutrition and sexual development, unilateral or bilateral primary optic atrophy, deformity of the sella turcica shown by the X-ray and proven changes (tumor, gumma, etc.) of the pituitary body at operation or autopsy. In fact, the statement has been made (6) that there is no record of necropsy in which the pituitary was examined and found normal.

Recently, however, Bailey and Bremer (7) have brought forth evidence to show that experimentally a lesion in the para-infundibular region of the hypothalamus, with careful avoidance of injury to the pituitary body, is followed by the characteristic polyuria.

Treatment has been largely symptomatic. Some cases have yielded to active antisyphilitic treatment, probably establishing this as their etiological factor. The quoted case of Herrick (3) has not been duplicated in the literature. Farini (8) demonstrated that subcutaneous injections of pituitary extracts would temporarily control the disease. But Osler (9), as late as

his eighth edition, does not mention pituitrin. This effect of pituitary extracts has been confirmed by many observers (6, 10, 11, 12, 13), and Blumgart (14) has since shown that the pituitrin can be given intranasally, or in salol coated pills, (Rees, 15), although when given directly by mouth it produces no effect.

The combination of pregnancy and diabetes insipidus is mentioned by French (16) who says that it "may occur." The combination of the acute retention of pregnancy and diabetes insipidus is not mentioned in the available literature and is the unusual feature of the following case.

Recently, acute retention in fevers, or postpartum or postoperative, has been treated by Stater (17) with benzyl-benzoate, theoretically to relieve the spastic condition of the vesical sphincter. The logic of its use has been confirmed experimentally in rabbits (18). Its use in this case was successful in relieving the retention, though it possibly was of importance in the abortion following.

The case is as follows:

An American housewife of 28 was admitted complaining of lower abdominal pain. The family and past histories were essentially negative. There was no history of similar disease in any of the members of her family.

She has had no illnesses since the childhood diseases and the influenza in 1918. She habitually drinks large quantities of water, "bucketsful," as she expresses it, and passes large amounts of urine. She has had two normal deliveries in the hospital in the past three years and has never passed urine of a specific gravity higher than 1,008. At one time in her last puerperium, she ran a slight temperature and a catheterized specimen of urine showed a small amount of pus. After the administration of urotropine the temperature dropped and the urine cleared.

Her last regular catamenia was 3 months before admission and she had had some of the usual subjective symptoms of pregnancy. Four days before admission she began to have increased frequency of urination and lower abdominal pain. She was catheterized twice and large amounts obtained. She became increasingly uncomfortable and came to the hospital.

General physical examination at admission was practically negative. She was well developed and nourished. There was no limitation of the visual field and the optic discs were normal. The blood pressure was 120-70. Pelvic examination showed a marital introitus, normal urethral meatus, softened lacerated cervix, and a fundus enlarged to the size of a normal 3 months' pregnancy in good position freely movable. The adnexa were not felt. Catheterization was accomplished without difficulty and removed an ovoid tumor felt in the mid line, yielding 3,250 cubic centimeters.

The leucocyte count was 10,400, with 49 per cent polymorphonuclears, 43 per cent small lymphocytes, 6 per cent large lymphocytes and 2 per cent transitionals. The red cells were normal. The blood Wassermann was negative to both plain and cholesterinized antigen. The blood sugar was 110 milligrams, and the blood urea nitrogen 12.8 milligrams in 100 cubic centimeters. The urine showed no sugar or albumin and varied in specific gravity between 1,003 and 1,006. The specific gravity of the blood was 1,065.

On a restricted intake, just consistent with bodily comfort and not sufficient wholly to satisfy the thirst, the output ranged between 5,000 and 8,000 cubic centimeters. For the first few days she was catheterized every 4 hours, voiding normally only in small amounts.

She was given 0.3 grams of benzyl-benzoate in capsules twice a day for a week and voided normally. This was discontinued and she required catheterization once, and being put back on the drug, resumed control of the sphincter. It was again discontinued, but 3 days later, though voiding normally, she developed lower abdominal cramps, began to flow, and after a few hours, discharged a 3 months' fetus. The fetus was normal in development and showed no stigmata or signs of disease.

After the abortion, she had no further retention although she continued to drink large amounts and to pass large quantities of urine.

X-rays show a slight anterior bending of the posterior clinoid processes. Temperature, pulse, and respiration were normal. Weight was constant.

On discharge, she was perfectly comfortable, with a urine of specific gravity 1,004.

In this case, there was no general, focal, or neighborhood signs of disease of the pituitary. The administration of pituitrin was considered but rejected for fear of inducing abortion. There was no local cause for the retention discovered except the pregnant uterus.

The urine at all times was low in specific gravity and the closest watch was necessary to prevent the patient from adding to her intake from unmeasured sources. With an output of 5 to 8 liters, she resorted frequently to washing her mouth with water, to relieve the thirst.

The retention itself was controlled by the administration of benzyl-benzoate, but whether or not it relaxed the smooth muscle of the uterus, and thus was a factor in the production

of the abortion, is uncertain. The large intake and output of dilute urine continued after the abortion and seemed wholly unrelated to the pregnancy, although there was no further retention following the termination of the pregnancy and the return of the uterus to normal size.

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HERNIA THROUGH THE FORAMEN OF WINSLOW

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INTESTINAL herniæ through the foramen of Winslow are extremely rare, only thirty cases verified either by operation or autopsy being found in the literature. They are as follows:

1. Blandin, in his second edition of Traité d'anatomie topographique ou anatomie des régions du corps humain, 1824, p. 467, reports an unexpected finding at autopsy. Almost all the small intestines had passed into the lesser sac through the foramen of Winslow. Through an abnormal opening in the transverse mesocolon they had reentered the general abdominal cavity and had become strangulated in this abnormal orifice.

2. In Rokitanski's Handbuch der speciellen pathologischen Anatomie, 1842, iii, 218, we find the statement: "I once saw a large portion of the small intestine strangulated by the opening of the foramen of Winslow."

3. Treitz, under Hernia retroperitonealis, in ein Beitrag zur Geschichte innerer Hernien, Prag, 1857, p. 126, gives a detailed description of an autopsy on a woman, aged 32, in which, among other abnormalties (absence of duodenum), two loops of jejunum were loosely caught in the foramen of Winslow, the edges of which were thickened.

4. T. Wilson Moir reports a case cited by Chiene in the Journal of Anatomy and Physiology, 1868, ii, 218, in which the patient had died of intestinal obstruction. At autopsy all the intestines were found to have passed into the lesser sac.

5. Novello, in Nel No. 38 della Gazetta medica delle provincie de Venete, Annuatio delle science mediche dell'anno 1881, cites a case reported by Majoli. At autopsy a loop of small intestine was found to have passed through the foramen of Winslow as the result of abdominal pressure.

6. Majoli, in Rivista clinica di Bologna, 1884, 605, gives an interesting personal obervsation followed by autopsy, the first case with clinical and anatomical data combined. A man, aged 42, after showing signs of obstruction, died on the seventeenth day of his illness. At autopsy the transverse colon and part of the greater omentum were found in the lesser sac. It was not possible even at autopsy to reduce the hernia with simple traction. An incision of the edges of the foramen had to be performed before the release of the strangulation became possible.

7. Elliot Square, in Brit. M. J., 1886, i, 1163, mentions the case of a man, aged 20,

with acute excruciating pain in the epigastrium, associated with vomiting, who was seen on the third day of his illness. Tenderness was found in the epigastrium. There was no bowel movement for four days. He died on the third day after the onset of symptoms. Autopsy: The intestines were distended; eight inches of ileum, two feet from the cæcum were incarcerated through the foramen of Winslow and were withdrawn with difficulty. The edges of the foramen were thickened and congested.

8. F. Treves, in Lancet, Lond., 1888, ii, 701, gives what is apparently the first case reported of a laparotomy with a hernia through the foramen of Winslow. A man, aged 26, developed symptoms three hours after a heavy meal. An operation for intestinal obstruction was done eight days after the onset of symptoms. Two or three feet of small intestine were reduced, but reduction of a second loop was impossible. It was not possible to enlarge the foramen without cutting the portal vein, hepatic artery and common duct. The patient died six hours later. Autopsy: The two or three feet of ileum that had been reduced looked purplish. The cæcum, ascending colon and part of the transverse colon had passed through the foramen. At the splenic flexure there was a sharp kink which accounted for the distention beyond the strangulations. Some peritonitis was present.

9. Gangolphe. Lyon méd., 1890, lxiv, 607. A soldier, aged 56 years, showed signs of acute obstruction. Forty years previously, he had had a similar attack of intestinal obstruction which had disappeared. At operation on the fourth day, a certain resistance which gave easily was found and on the appearance of a loop of intestine, which was grooved, the strangulation was believed to have been released. But at autopsy, 3 days later, 1.5 meters of ileum was found in the lesser sac. The intestines had perforated in places and fæcal material lay free in the lesser sac.

10. Rehn. Arch. f. klin. Chir., Berl., 1892, xlviii, 310. A diagnosis of acute intestinal obstruction was made on a man aged 77. Laparotomy was performed on the third day of the illness. With firm traction a loop of gut, 15 centimeters in length, was withdrawn from behind the stomach; it presented evidences of obstruction but the intestine was in good condition. Death was probably due to an overdose of opium.

11. Neve, Arthur. Lancet, Lond., 1892, 1175. This is the first case of a cure after surgical intervention in a male (aged 17) with incomplete obstruction. Laparotomy was performed 28 days after the onset of symptoms. The hiatus, through which had penetrated part of the transverse colon, admitted two fingers. Other loops of intestine could not be withdrawn without tearing them. Traction failed. In spite of these findings the patient recovered.

12. Picado, J. S. Revista de la Sociedad médica argentina, Buenos Aires, 1893, ii, 10. A soft tumor had appeared in the epigastrium of a boy aged 8 years, ten days before the onset of symptoms. Symptoms of acute obstruction developed. A laparotomy was done on the twenty-first day, but the patient died. Autopsy: The distal end of the ileum was extracted with difficulty from the lesser sac. The ascending colon was invaginated into the transverse colon for 20 centimeters.

13. R. Stecchi. Clin. chir., Milano, 1894, ii, 653. The case occurred in a male, aged 63 years, with epigastric pain, distended abdomen, and vomiting. Enemata were ineffectual. A laparotomy was performed on the third day. The small intestines were distended; the large intestine flaccid; the foramen of Winslow was dilated sufficiently to admit the entire hand. The patient died on the third day after operation. Almost the entire small intestine was herniated into the lesser sac. The omentum was adherent to the inferior border of the foramen of Winslow producing the angulation of the transverse colon near the splenic flexure and thus causing the obstructive symptoms.

14. Reynier. (Quoted by Jeanbrau and Riche.) At an operation upon a patient, who was *in extremis*, there was found a hernia of small intestines through the foramen of Winslow. The gangrenous gut was reduced. Death occurred on the same day. No autopsy.

15. Mori, G. Gazz. med. lomb., Milano, 1898, vii, 257. There were signs and a history

of acute obstruction in a man, aged 50. A laparotomy was done on the fifth day. The small intestines were distended. The cæcum and ascending colon were not seen; the descending colon, sigmoid, and rectum were empty. By exclusion, the diagnosis of hernia through the foramen of Winslow was made. Reduction by traction was performed. The patient had a normal stool immediately after the operation.

16. Groves and Marten. Indian Medical Record, 1901, xx, 333. There were signs of acute intestinal obstruction in a woman aged 47 years. Laparotomy was done on the fifth day. The transverse colon was engaged in the foramen of Winslow, through which two fingers could be easily passed. The intestine was reduced easily by traction. An enterotomy for the relief of distention was done on the lesser bowel, and a cæcostomy was also performed. A fæcal fistula developed and closed later. The patient recovered.

17. Delkeskamp. Beitr. z. klin. Chir., 1905, xlvii, 644. The patient was a female, aged 22. Immediately after a normal labor she was taken with severe pains in the epigastrium. The abdomen became distended, vomiting occurred, and no flatus and fæces were passed. At operation, seven days after the onset of symptoms, 8 centimeters of ileum and almost the entire large intestine were seen passing through an opening which admitted three fingers. This was believed to be the foramen of Winslow. These intestines were covered with a peritoneal fold which proved to be the anterior sheet of the lesser sac. This was torn into, some adhesions broken up, and the herniated intestines drawn into the abdominal cavity. The patient was cured.

18. Jeanbrau and Riche. Rev. de chir., Par., 1906, xxxiii, 618. The patient was a boy, aged 6, with signs of acute intestinal obstruction, who was seen on the third day of his illness. An epigastric swelling was present. At a laparotomy, the cæcum, large intestine and part of the small intestines were found collapsed. The duodenum, jejunum, and major part of the ileum were distended. The cause of obstruction was not determined, but was believed to be strangulation through the foramen of Winslow. An enterostomy was done for fear of tearing into the intestines. The patient developed symptoms of meningitis and died. No autopsy was obtained.

19. Rawitsch-Schtscherbo. X Jahresb. f. Chir., 1900, iii, 627. A diverticulum of the small intestines had passed through the foramen of Winslow. No operation was done but autopsy revealed the condition.

20. Adjaroff. (Quoted by Hilgenreine. Prag. med. Wchnschr., 1903, 571.) The small intestines were herniated through the foramen of Winslow. The patient died soon after operation.

21. Morton, Charles A. Brit. Med. J. 1909, l, 641. Immediately after defecation there occurred symptoms of acute intestinal obstruction. A laparotomy was done seven hours after the onset of symptoms. Intestinal loops were withdrawn from what corresponded to the foramen of Winslow. The small intestines were evacuated with a Paul tube which was sutured to the outside. There was leakage around the tube into the peritoneal cavity. Death occurred one week later from general peritonitis.

22. Cawardine. Lancet, Lond., 1909, ii, 1215. An engineer, aged 44, was taken with violent abdominal cramps while he was telephoning. Vomiting of bile occurred two days later. Rigidity was present in the epigastric region. An operation was done on the second day after the onset of symptoms. The patient was almost pulseless. Some of the intestines were reduced, but a vascular band prevented complete reduction. Gangrene was present. An enterostomy was performed. The patient died three hours later. An autopsy was obtained.

23. W. Haw. Lancet, Lond., 1909, l, 1598. A colored boy, aged 5, was seen in a moribund state and died a half hour later. Autopsy: The cæcum, appendix, and part of the ileum had passed through the foramen of Winslow. Reduction of the hernia was impossible without dividing the edges of the orifice and injurying the vessels. The cæcum and appendix were filled with worms.

24. E. Schwalbe. Virchow's Arch. f. path. Anat., etc., 1904, cxxxvii. This is a report of an autopsy. A woman, aged 34, died of parenchymatous nephritis and aortic and

mitral insufficiency. The small intestines in the region of the duodenojejunal angle had passed into the lesser sac without being strangulated by the edges of the foramen of Winslow.

25. Haymann. Thesis, Munich, 1892. The patient, aged 20, with signs of pulmonary tuberculosis, was brought into the hospital. The following day there developed symptoms of severe acute obstruction which suggested a high obstruction. A laparotomy with median incision, above and below the umbilicus was done. The operator plunged his hand into the region of the duodenojejunal fossa and the hernia was immediately reduced. It was thought that the intestines had herniated through the foramen of Winslow. The patient died a quarter of an hour after intervention. At autopsy the stomach was dilated; pulmonary tuberculosis was present.

26. Thomas Sinclair. Brit. M. J., 1909, I, 645. A man, aged 58, developed abdominal symptoms suggestive of acute intestinal obstruction. Following a spell of coughing there was fever and an epigastric swelling. A laparotomy was done on the third day. A mid line incision above the umbilicus was employed. The foramen of Winslow tightly constricted the intestine. The edges of the orifice were dilated gently and 2½ feet of jejunum were then withdrawn with ease. An annular constriction, almost gangrenous, was present. This was fixed to the parietal wall and the free border of omentum was carried around it, so that, if rupture did occur, it would take place into the omental sac. No complications occurred and the patient was cured.

27. Radovan. Des hernies etranglées de travers de l'hiatus de Winslow, 1919, Thèse de Paris. A woman suddenly developed excruciating pain in the right iliac fossa. For the first few days it was intermittent but later constant in character. Very little vomiting occurred and fæces and flatus passed. Seven days after the onset, the condition of the patient became critical. Just before that a tumor had appeared in the upper abdomen, and pain persisted in the right iliac fossa.

An operation was done with a mid line incision above and below the umbilicus. Below the stomach, there was a rounded mass under tension which suggested a lobulated cyst. A flaccid loop of intestine, which lost itself in an orifice behind the hepatic pedicle, was picked up. Passing out from this same orifice, some dilated small intestine was seen. It was impossible to reduce either loop by traction. An incision was made through the transverse mesocolon; and an enterotomy for the relief of distention and evacuation of the intestinal contents was done and closed up immediately. After this, reduction became possible. A great part of the transverse colon, all of the ascending colon, cæcum and appendix and a small part of ileum were incarcerated in the lesser sac, and had formed the globular mass already described. Cured.

28. J. E. Engstadt. J. Am. M. Ass., 1919, lxxii, 411. Sudden pain in the epigastrium was noted while lifting a heavy object twelve hours previously. The abdomen was rigid and tender. A tense mass was present in the epigastric region. Laparotomy: The upper portion of jejunum was strangulated through the foramen of Winslow. Traction was useless for reduction of the hernia. It was found necessary to introduce the tip of the little finger into the tight opening, carefully severing first the peritoneal coat and gradually the connective tissue of the opening. Great care was taken not to injure the portal vein or common duct. After this, moderate traction accomplished reduction. Two small drains were employed and were removed on the second day. A fæcal fistula developed on the tenth day, which discharged for five weeks and finally closed.

29. Schmilinsky. Deutsche med. Wchnschr., 1919, xlv, 4. A man, aged 65, for three weeks had had pains in the epigastrium after eating. Laparotomy: The cæcum and last part of ileum had entered the lesser sac through the foramen of Winslow. These had torn the lesser omentum and lay free just above the lesser curvature of the stomach. Traction was employed and reduction was readily accomplished. Flatus and fæces passed on the second day after operation.

The following case brings the series of herniæ through the foramen of Winslow up to 30.

May 27, 1922, T.D., a man, aged 49, was admitted as an emergency case. He was toxic and no history could be obtained from him. From his family physician it was learned that he had been taken ill one week previously with general abdominal pain, nausea and vomiting. For the first two or three days, constipation was present. Later a severe diarrhœa developed. The patient grew progressively worse. He had refused to come to the hospital for about a week. Marked distention of the abdomen was present for two days before he was admitted. No history of any previous digestive or abdominal disturbances was obtainable. The past history was negative, except for furunculosis in 1920.

P. E. Middle-aged man, eyes sunken, face pinched and haggard, skin clammy; very toxic; orientation poor; head negative; heart and lungs negative. *Abdomen.* Distention throughout, but more marked in upper half. No peristaltic waves seen. Marked resistance was present but very little tenderness. Tympany was present throughout. Movable dullness was noted in the flanks. No masses were made out. Urine: positive for albumin and hyaline casts. Blood: white blood cells, 16,400, 86 per cent polymorphonuclears.

Operation. Under gas and ether anæsthesia a right rectus incision was made. Brownish fluid escaped on opening the abdomen. The small intestines were greatly distended. Below the cæcum, the intestines were flaccid. In the presence of the distended loops, the point of obstruction was not determined. Several punctures were made for the escape of feces and flatus, but the distention was only partly relieved. These enterotomies were closed with black silk. The patient's condition was so critical that it was thought best to do as little operative manipulation as possible. A loop of distended gut was brought out of the abdomen and sutured to the peritoneum. This was later opened with the cautery. Patient died about six hours later.

Autopsy. The operative incision was enlarged up and down. Almost all of the small intestines were dilated. The large bowel was collapsed. Four inches of ileum, one and one-half feet from the ileocæcal valve had passed through the foramen of Winslow. This was withdrawn without difficulty from the lesser sac. This portion of gut was almost gangrenous, reddish-black in color.

The enterostomy was performed two feet above the obstruction.

ANATOMICAL CONSIDERATIONS

The foramen epiploicum, or the foramen of Winslow, was first described by Winslow in 1776. It is a semilunar or semicircular orifice which serves as a communication between the lesser sac and the general peritoneal cavity. It is about 8 centimeters in circumference, 5 centimeters from the mid line on the right at the level of the upper edge of the first lumbar vertebra. It corresponds on the abdominal wall to the intersecting point between a line uniting the seven costal cartilages and a vertical one, one finger's breadth external to the right border of the sternum. Studies on the cadavers by Jeanbrau and Riche have shown that the orifice easily admits the index finger. Radovan in his researches, also on cadavers, found that the index and middle finger could be easily introduced. The foramen of Winslow is bounded anteriorly, by the lesser omentum; posteriorly, by the inferior vena cava; above, by the caudate process of the liver; and below, for all practical purposes, by the first portion of the duodenum.

The lesser omentum should be considered in connection with the surgery of the foramen of Winslow. It forms the anterior boundary of the hiatus. Between the two layers of the peritoneal folds of the hepatoduodenal ligament, the portal vein can be found behind with the hepatic artery and common duct in front, the former to the left and the latter to the right of the portal vein. The edges of the orifice cannot be incised without injury to these three structures.

Jeanbrau and Riche in a splendid article have called attention to a relatively bloodless space in the posterior leaf of the gastrohepatic ligament—the inter-porto-choledochus space. It is triangular in shape with the base below and mostly retroduodenal. In the region of the pancreas and the duodenum, the interval between the portal vein and common duct is quite large and allows one to enter the lesser sac without injury to the above structures.

ETIOLOGY AND MECHANISM IN THE PRODUCTION OF THE HERNIA

Herniæ through the foramen of Winslow are rare, no doubt due to the fact that the orifice is placed high in the abdominal cavity and is concealed by the small intestines and the transverse colon. Violent efforts may be responsible and explain the greater frequency in men. Only five females are mentioned (Treitz, Groves and Marten, Delkeskamp, Schwalbe and Radovan) as compared to 20 males, and in 5 no sex is mentioned.

The following occupations are noted: gardener (Rehn); agriculturist (Mori); engineer (Cawardine).

In one case, the onset of symptoms began immediately after a difficult stool (Groves and Marten) and in another, after defæcation (Morton); after a coughing attack (Sinclair); and immediately after a normal labor (Delkeskamp); two hours after a heavy meal (Treves); and while lifting a heavy load (Engstadt).

Any undue abdominal contraction, as in constipation, defæcation, labor, or heavy lifting may be a factor in the production of a hernia through the foramen of Winslow. It is possible that purgatives increasing peristaltic movements may be another agent in its etiology. An existing obstruction may be responsible; the intestine combating against the obstruction and trying to empty itself may enter the foramen, as in Picado's case.

AGE

The age at which hernia through the foramen of Winslow occurred is indicated in 20 patients. The youngest was 5 years and the oldest 66 years. This type of hernia may occur at any age. The following ages are noted: 5 years (Haw); 8 years (Picado); 6 years (Jeanbrau and Riche); 20 years (Hayman); 22 years (Delkeskamp); 25 years (Square); 26 years (Treves, Neve); 32 years (Treitz); 34 years (Schwalbe); 44 years (Majoli, Cawardine); 46 years (Groves and Marten); 49 years (Ullman); 50 years (Mori); 56 years (Gangolphe); 58 years (Sinclair); 61 years (Stecchi); 64 years (Rehn); 65 years (Schmilinsky).

CAUSES OF STRANGULATION

The strangulation of the bowel is, as a rule, the result of constriction by the edges of the orifice, which is often thickened and congested. Jeanbrau and Riche have described a peritoneal fold at the lower edge of the foramen and attributed to it an important rôle in the causation of the strangulation. In Stecchi's case the symptoms of obstruction were due to an adhesion of the greater omentum to the edge of the foramen, and not to strangulation at the orifice. In another case (Blandin), the intestines passed through a dilated foramen of Winslow and the obstruction was caused by a narrow abnormal opening in the transverse mesocolon. It is well to bear in mind that intestines may pass through the foramen of Winslow without being strangulated.

CONTENTS

The hernial contents are variable lengths of small or large intestines and occasionally the greater omentum (Majoli, Stecchi). The small intestines are found herniated into the lesser sac twice as often as the larger bowel. In twenty-seven observations, nine were cases in which the large intestines had passed through the foramen of Winslow: (1) transverse colon (Groves and Marten); (2) Neve; (3) Majoli; (4) cæcum and ascending colon (Mori); (5) cæcum, ascending colon, and part of transverse colon (Treves); (6) end of ileum, ascending colon and cæcum; (7) appendix, cæcum (Delkeskamp), and part of ileum (Haw); (8) ileum, appendix, cæcum, ascending colon and greater part of transverse colon (Radovan); (9) cæcum and last part of ileum (Schmilinsky).

In three, almost the entire length of the small intestines passed through the foramen of Winslow (Blandin, Stecchi, Reynier).

SYMPTOMS

The symptoms of strangulated hernia through the foramen of Winslow are those of intestinal obstruction; namely, pain, vomiting, distention, absence of fæces, and flatus.

Clinical studies follow, based on twenty-two cases of the total thirty.

Pain. As a rule, the site of the pain is in the epigastrium or the umbilical region, and occasionally in the right hypochondrium. Often there is generalized abdominal pain (Cawardine, Ullman). In Haymann's patient with advanced tuberculosis lesions, pain was localized to the lower right thorax. In one, the pain was referred to the right iliac fossa (Radovan).

As a rule, the onset of symptoms is sudden. In some cases (Picado, Groves and Marten, Schmilinsky, Ullman), the symptoms progressed gradually and suggested incomplete obstruction. The acuteness of onset depends

on the degree of intestinal constriction. At times, the pain may be terrific. Square's patient doubled up; in Treves' patient, the pain was so severe that the patient could not sleep and had to pass the night in a chair. Radovan described his patient as having violent cramps.

Vomiting. Vomiting is an early sign. But in Sinclair's case it did not appear until the third day of the illness. At first, it is liquid in character; later it becomes fæcal. Yet in Majoli's case, where the obstruction was not complete until the thirty-fifth day, vomiting was fecal from the start. In many, the type of vomiting is not indicated; some speak of frequent vomiting. In Treves' case, the vomitus was always intestinal; in Jeanbrau's and Riche's case, vomiting was alimentary at the time of operation. Cawardine's patient had vomitus as black as ink. Morton also described the vomitus as biliary in character.

Absence of fæces and flatus. This is the most important of all signs and is almost always constant. In Majoli's and Neve's cases, the obstruction was never complete and in both the large bowel was strangulated. In my case, constipation was present for two or three days and was followed by a severe diarrhœa up to the time of the operation. But in most of the cases nothing passed by the bowels, enemata were ineffectual and the obstruction was complete from the start.

Physical signs. An important sign is an epigastric or perimubilical swelling appearing soon after the onset of symptoms. As a rule, the swelling is in the mid-line, though occasionally it encroaches on the right hypochondrium. Even in the presence of generalized abdominal distention, the tumefaction in the epigastric or umbilical regions stands out prominently. But, as a rule, the tumor is masked by the abdominal fullness. It is important, therefore, to examine the patient early before the distention has become general. Epigastric swellings are noted by the following: Majoli, Neve, Picado, Stecchi, Mori, Jeanbrau and Riche, and Sinclair. Cawardine described the abdomen as distended, but offering resistance in the epigastric region. Engstadt's patient also had abdominal distention with a tender mass in the epigastrium. In my case, the entire abdomen was ballooned out, but the distention was more marked in the upper half. Periumbilical swellings were noted in two cases (Reynier, Square). In others, nothing more than abdominal distention was made out. These tumefactions, the epigastric and periumbilical are present, irrespective of the portion of the intestine strangulated; in Picado's and Stecchi's cases, it was the small intestine; in Majoli's case it was the transverse colon.

DIAGNOSIS

A pre-operative diagnosis of a hernia through the foramen of Winslow has never been made, and rarely has it even been suspected. Neve was the closest with the diagnosis of internal hernia. Intestinal obstruction is the usual diagnosis.

Even at operation the diagnosis has often been missed, especially in the presence of distended loops of intestine. Treves and Mori arrived at a diagnosis by a process of exclusion. Stecchi thought of the condition after he had reduced the hernia and introducing his hand into the foramen of Winslow found that it was easily admitted. Haymann also suggested a hernia through the foramen of Winslow but never was able to prove it. Positive diagnoses at operation, however, were made by Sinclair, Radovan, Engstadt and Schmilinsky. In my case, the patient was so toxic that no effort was made to learn the cause of the obstruction. An enterostomy was the only operative procedure justified.

TREATMENT

Surgical intervention is an immediate indication when intestinal obstruction is suspected. Were it possible to make a diagnosis of a hernia strangulated through the foramen of Winslow, the incision would no doubt be the same as for any biliary operation. In most of the cases a mid line incision, extending above and below the umbilicus for variable lengths was employed, often with excellent exposure.

Twenty laparotomies were done; twelve of these were followed by death and eight patients were cured. The remaining ten were either autopsies or cases in which death

occurred before any operative procedure was attempted.

If the patient is desperately ill, it is wise to do as little as possible—an enterostomy on the first distended loop that presents itself. More, of course, can be attempted in patients who are better risks. If possible one should determine the cause of the obstruction: (1) adhesions, (2) volvulus, (3) torsion of mesentery, (4) intussusception, (5) strangulation in an abnormal internal orifice of the mesocolon or the mesentery or in the duodenojejunal fossa. To determine if the hernia has passed through the foramen of Winslow, the index finger should be introduced under the liver and search be made along the right border of the gastro-hepatic omentum to make out the pulsation in the hepatic artery.

If one is reasonably certain he is dealing with a hernia strangulated at the foramen of Winslow, he may then proceed with the following operative manipulations:

1. *Reduction by simple traction.* It is possible that reduction may occur as soon as the operator plunges his hand into the abdomen (Haymann). In certain cases (Mori, Groves and Marten, Delkeskamp) the herniated bowel was easily withdrawn from the lesser sac simply by traction. In others (Rehn) it was reduced by this method with difficulty. In others, again (Treves), traction failed to release the strangulation, and in Majoli's case this was impossible even at autopsy. Cawardine found that complete reduction by traction was prevented by a vascular band and that only partial reduction could be accomplished.

2. *Preliminary enterotomies and traction.* Radovan has emphasized initial preliminary enterotomies, single or multiple, followed by careful closure of the intestine with a double row of Lembert sutures. These enterotomies are performed in order to lessen the distention and to evacuate the bowel, thereby facilitating reduction by simple traction of the hernia strangulated through the foramen of Winslow.

Two points are to be observed where simple traction is employed. (a) See to it that every loop of strangulated intestine has been disengaged. Introduce the finger into the foramen of Winslow to determine if it is empty. Gangolphe thought he had liberated the entire hernia when he saw a point of obstruction; but at autopsy more than a meter of intestines was found in the lesser sac. (b) See to it that the greater omentum is not in the lesser sac. In Stecchi's case, the hernia was reduced, but at autopsy, three days later, the omentum which was adherent to the edge of the foramen had caused angulation of the transverse colon and had given rise to intestinal obstruction.

3. *Reduction by traction after dilatation of the Foramen of Winslow.* If with gentle traction the strangulation does not yield, strong traction is contra-indicated. The intestines have already degenerated to some degree and violent methods may rupture the bowel. One may try to dilate the foramen, employing the utmost care.

In this series of cases, two fall into this group (Sinclair and Engstadt). The former applied gentle traction to the orifice and then found that the herniated intestines could be withdrawn from the lesser sac with ease. In the latter case, it was found necessary to introduce the tip of the little finger into the tight opening, carefully severing, first, the peritoneal coat and gradually the connective tissue of the foramen, great care being taken not to injure the portal vein and common duct. Reduction was then rendered possible with moderate traction.

If the methods just described prove unsatisfactory, there remain two procedures at the disposal of the surgeon: (1) reduction of the hernia after opening the lesser sac and (2) reduction after incising the orifice (*débridement*). Opening into the lesser sac is the method of choice, first, because incision of the orifice may injure the portal vein and common duct, and secondly, opening into the lesser sac allows for inspection of the herniated contents to determine the condition of the loop of bowel, whether adhesions are present or whether the intestines are twisted or perforated. Through such an opening it is possible to perform preliminary enterotomies upon the strangulated intestine for relief of distention and the discharge of intestinal contents. Blandin found that the intestine had passed through the foramen of Winslow without

strangulation, and had then passed through an abnormal opening in the transverse mesocolon and was strangulated there. In Schmilinsky's case, laparotomy revealed a hernia through the foramen of Winslow which had bored into the lesser omentum; the cæcum and last part of ileum lay just above the lesser curvature of the stomach. Taxis was employed and the hernia yielded without difficulty.

In Delkeskamp's case, almost the entire length of the large intestine was seen to have passed through the foramen of Winslow, which was dilated to admit three fingers. The intestines were in places distended and in places flaccid and were covered with a peritoneal fold which proved to be the anterior sheet of the lesser sac. This was torn open, some adhesions were released, and the intestines were then drawn into the abdominal cavity through the foramen of Winslow.

In Radovan's case, the lesser sac was opened through the transverse mesocolon, the intestines emptied by enterotomies and reduction accomplished by traction.

Whether to enter the lesser sac through the lesser omentum or the transverse mesocolon is a question of judgment for the surgeon, but the latter approach is regarded as the safer.

Enlargement of orifice by incision. This method has never been tried on a human being. Treves and Neve were in accord in their views of the impossibility of this method. Incision of the edges is dangerous. The hepatic artery, common duct and portal vein are liable to injury. Jeanbrau and Riche have elaborated a technique of *débridement* on the cadaver by approaching the foramen through a route which they call the inter-porto-choledochus space. This lies behind the pancreas and is retroperitoneal. Here the interval between the portal vein and common duct is quite large and allows easy access into the lesser sac. For detail the reader is referred to the original article.

This method appears difficult and time consuming, and is not a proper operative procedure where speed is an important factor. Silvada Braner called attention to the dangers associated with it. In twenty cadavers the space did not exist and in the remaining half it was traversed by various blood vessels.

In conclusion, one might ask, will a preoperative diagnosis ever be made of a hernia strangulated through the foramen of Winslow and will it be differentiated from other internal herniæ? That remains to be seen. At present, the important point is to *diagnose intestinal obstruction and to operate at once.*

In this way the prognosis in herniæ strangulated at the foramen of Winslow can no doubt be rendered more favorable.

DEPARTMENT OF TECHNIQUE

COMPRESSION LEVERAGE FRACTURES OF THE ANKLE JOINT

By JAMES H. STEVENS, M.D., Boston, Massachusetts

THE accepted classical definition of a Pott's fracture is a fracture of the fibula 2 to 4 inches above the joint, and an accompanying fracture of the internal malleolus or a ruptured internal lateral ligament. Pott described a fracture of the fibula 3 inches above the joint and a broken internal lateral ligament and this is so rare as to be negligible, a rupture of the internal lateral ligament occurring, in any of these various fractures as we shall prove, only as the result of extreme dislocation of the astragalus. We are told that all the other fractures of the fibula at its lower end are not Pott's, because there is no breaking of the internal malleolus and no rupture of the internal lateral ligament. One could easily imagine a fracture of the fibula by a comparatively slight blow (direct violence) which would produce nothing except a bony lesion. The damage would be slight and the prognosis, in such a fracture would always be good.

But a man steps from a curbing, his foot strikes an uneven surface in the pathway and turns outward. There is impact behind the force, and the weight of the man is added to the impact. The fibula breaks at the joint surface, usually, but occasionally above it, as in the classical type.

The inferior tibiofibular ligaments are stressed, always damaged, the tip of the fibula swings outward, and usually backward. There is widening of the mortise of the ankle joint and whether or not it occurs, there is the potentiality of dislocation. There is a great deal more damage than in the case of the direct blow, and there is a tremendous injury to the joint itself which is not present in the other case. One is ephemeral. The other is dynamic, and its effects are lasting.

There is strikingly little difference in the X-rays to the casual observer, but the difference is there. In disagreement with many of my colleagues, if we are to cling to the old nomenclature, such an injury is a Pott's. If the ordinarily accepted definition is only a definition of one variety of the fracture and that variety one which, while more severe, is numerically less than other types and if by the very specificity of its wording, it excludes absolutely the others, then it is clear that it has not only failed as a definition but it is infinitely worse than this because it has befogged the whole subject.

If we classify these injuries under the terminology which we have used in this paper, we shall be able to group them scientifically since etiologically there are certain mechanical features which are common to all.

First, compression, because it is the most important feature and it is always present in every case, the weight of the man (static compression) plus the infinitely greater compressive stress due to impact. These breaks at the ankle are not pure compression breaks, but the compression by keeping the astragalus firmly against the tibiofibular mortise, while at the same time causing the locking of all the tarsal articulations determines to a great extent the character of the injury.

Second, leverage, because there is a leverage stress in two directions with compression on one side of the breaking bone and tension upon the other.

We subdivide this into eversion and inversion, and by further subdivision we are able to group all these various types in a truly scientific manner which was never possible under the old nomenclature.

The etiology of all these injuries to the ankle joint is practically the same. A person steps from a height like a curbing and turns his ankle. He falls or slips and does the same

thing. Running or jumping he turns his ankle in alighting. In the case of ball-players he slides for a base and encounters an obstruction with his foot. Sometimes with a heavy person it happens in the course of ordinary walking over rough ground but usually there is the phenomenon of impact.

Now the medical profession, as a whole, is united on certain of the fundamental mechanical features of production while differing on minor points.

Stimpson, Bonnet, and Tillaux believe that the internal lateral ligament tears or the internal malleolus breaks first and is followed by a fracture of the fibula.

Meissonneuve, Speed, Ashhurst, and Murphy believe that the fibula breaks first and that following this break the internal lateral ligament tears or pulls off the internal malleolus.

So that there is agreement between these two groups, that there must be a turning downward of the astragalus. Because if the internal malleolus breaks before the fibula through the pull of the internal lateral ligament, as they claim, or the internal lateral ligament is torn, then this can be accomplished only by a dropping down of the inner edge of the astragalus, and equally as clearly if the fibula has broken first and the internal malleolus is broken by the pull of the internal lateral ligament or the ligament is torn as the other group claim, then it must also be done by a dropping down of the inner edge of the astragalus, because if caused by the astragalus slipping outward, there would always be the outwardly dislocated astragalus remaining, and this is not true. Many times the astragalus is not dislocated and nevertheless the internal malleolus is broken. This is a statement of fact and can admit of no controversy.

The third class, and this is composed of some of each of the other classes, believes that there is a rotation outward of the foot pivoted on the astragalus and that the internal lateral ligament breaks first or pulls off the internal malleolus. This was Meissonneuve's idea originally.

In order to demolish the theories of the two first groups, it is only necessary to prove that the astragalus does not turn and in order to demolish the contentions of the third group it is only necessary to prove that the internal lateral ligament does not tear and is not stressed except as the result of a dislocating astragalus. No one cares what a man's individual opinion may be if he cannot prove it. If he can, it will stand by itself even against a combination.

Figure 1 represents schematically the claim which is made by these two groups, that the astragalus is turning to bring the line A–D into the line A–D'. Can we prove this impossible?

Figure 3 will show you that the bones of the leg and foot constitute a column varying in area at different points and that the base of this column is offset to the fibula side always. In other words you walk on the fibula side of your foot.

Let us call the area of our column at the level of the astragalus as 1 inch by 1 inch (the bearing surface) and the man's weight 150 pounds. By the use of a simple mathematical formula it is possible to determine the compressive stress within practical limits. Let A equal the length and B the breadth of any section of our column at any level.

$$\frac{Load}{A \times B} = \text{Compressive stress in pounds per square inch;}$$

$$\frac{150}{1 \times 1} = 150 \text{ pounds of compression.}$$

This compressive stress is the same over the entire area of the astragalus. But this is not all. The center of compression of our column is the center of the column, the neutral axis. But because the base is offset in the ordinary standing position the line of resistance through the vase is fully 0.5 of an inch away from the center of compression and when the os calcis abducts under force, this line of resistance is further removed so that it may even fall outside the fibula surface of the column.

This last mentioned condition in the face of stress would mean infinity, it could not be measured.

But let us assume the problem as before with an offset of 0.5 of an inch. The offset is called the lever of eccentricity.

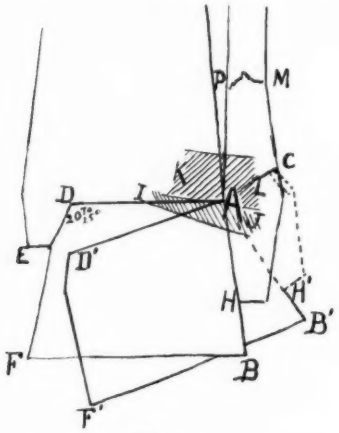

Fig. 1.

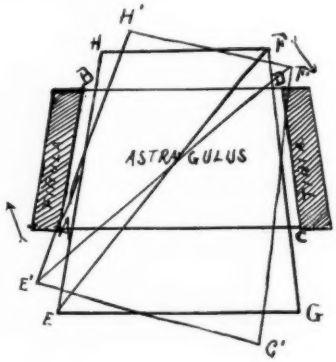

Fig. 2.

Fig. 1. This is a schematic representation of the movement to which is attributed the broken external malleolus and the supposedly ruptured internal lateral ligament by most surgeons. It is clear that if point *D* were to drop to point *D*¹ the external malleolus would be broken; *I. J.*, the transverse ligament would give way and often times, *K. L.*, the inferior tibiofibular ligament also. The internal lateral ligament would nearly always be ruptured. No such movement is possible as an examination of Figure 3 will clearly prove. The astragalus slides over against the external malleolus to exert pressure undoubtedly, but it cannot turn. The fibula breaks across *A. C.* and at times, as explained in the text, at *P. M.*, because subjected to three stresses at the same time.

Fig. 2. The rotation stress. This is a representation of the twisting stress or rotation stress due to the lever of rotation. The figure explains itself. Roughly speaking, a parallelogram is rotating to force its oblique distance between two rigid walls which are large enough only for one of its sides. It cannot be done and *A* and *D* will show the pressure points, and the arrows the direction of stress. Reversed it applies to inversion as well. This is the stress which breaks the internal malleolus so often in an everting foot and the external malleolus in an inverting foot, and such breaks are made possible only because of the compression which is illustrated in Figure 3.

Load $\times$ eccentricity $= \frac{F \times A \times B2}{6}$, in which

F is the cortical stress in pounds per square inch.

$$150 \times 0.5 = \frac{F \times 1 \times 1 \times 1}{6}$$

$F = 450$ pounds compression on fibula side;
$F = 450$ pounds tension on tibial side.

On the fibula side, we must add the 150 pounds of straight compression, and deduct it from the tibial side.

Stress in compression. Fibula side of our column at the astragalus—600 pounds per square inch.

Stress in tension. Tibial side at the astragalus—300 pounds per square inch.

Can the astragalus turn? Can 600 pounds be offset by 300? If it cannot then we have demonstrated our point. This is only the stress of common static load in walking. This disposes of the first two groups and their contentions.

We shall deal later with the third group when we consider the internal malleolar break.

If we apply the same reasoning to the fibula break and call our section 2 inches by 1 inch, reckoning the eccentricity only as before, the cortical stress developed over the fibula would be 300 pounds per square inch as against a tibial stress in tension of 150. Now add impact which we did not do in our previous problem and which is many times static stress (see Merriman *Mechanics of Motion*) and we shall begin to visualize what may happen when a man weighing 150 pounds steps from a 4 inch curbing and turns his

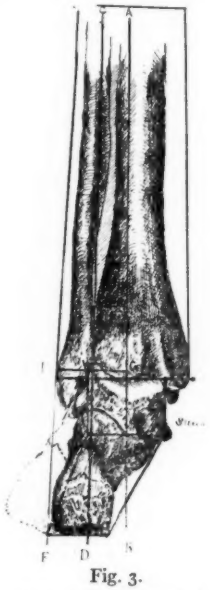

Fig. 3.

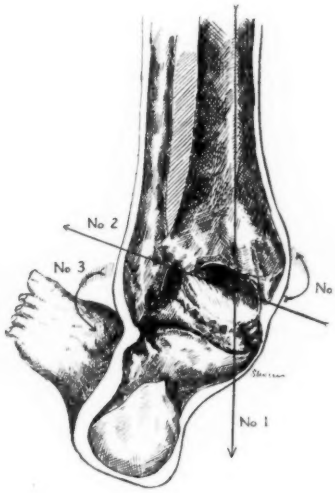

Fig. 4.

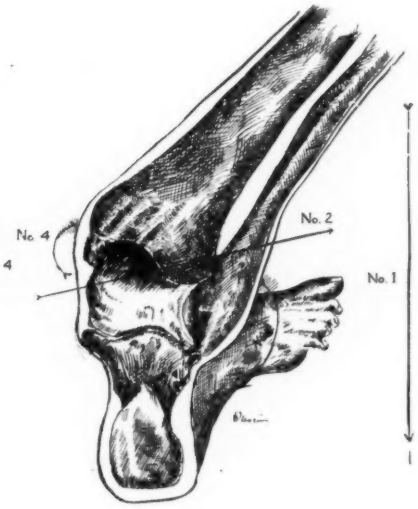

Fig. 5.

Fig. 3. The distance from the center of compression to the line of resistance in a column or strut is the lever of eccentricity. Problem: Weight of man, 150 pounds; area of column at astragalus, 1 inch by 1 inch; lever of eccentricity, 0.5 inch. *A.-B.*, center of column and also neutral axis in ordinary standing position of foot; *C.-D*, line of resistance; *G.-H.*, lever of eccentricity in abducting represented by dotted line; *F.-E.*, line of resistance; *G.-I.*, lever of eccentricity.

$\frac{150}{1\times1}=150$ pounds of straight compression, due to static load over entire column. Additional stress due to eccentricity.

$150\times0.5=\frac{F\times1\times1\times1}{6}$ in which F is cortical stress.

$F=450$ pounds compression on fibula side.
$F=450$ pounds tension on tibial side.

450 pounds compression due to eccentricity plus 150 pounds straight compression equals 600 pounds compression on fibula side at astragaloid level.

450 pounds tension due to eccentricity on tibial side minus 150 pounds straight compression equals 300 pounds. Tension on tibial side of our column at the astragaloid level.

Can 600 pounds be offset by 300 pounds?

If not, the astragalus cannot turn.

The heavy outside lines show how the leg and foot bones represent a column always with an offset base to the fibula side which explains why fibula fractures are more common and why the apparently simple cases are always serious injuries. Dissemination of great compressional stress results in much injury and is not confined to the bony structure.

Fig. 4. First method in mechanics of eversion. Arrow 1 shows center of gravity internal to everting foot. Arrow 2 shows resultant of the eversion stress due to the lever of abduction. Arrow 3 shows direction of the twisting stress, responsible for long fragments and for posterior displacement of the inferior fibula fragment, due to the lever of rotation. Arrow 4 shows direction of the twisting stress expended on the internal malleolus and causing not alone its fracture, but the anterior displacement of the internal malleolus so often present in the eversion type due to the lever of rotation.

Note in both drawings, Figures 4 and 5, the relations of the tip of the fibula to the os calcis at the astragaloid os calcis articulation showing how impingement of the articular rim of the os calcis in sliding outward, may break off the external malleolus low down when the stress is mild and the victim recovers his equilibrium in time to avoid a more serious injury.

Fig. 5. Second method in mechanics of eversion fractures: Arrow 1 shows center of gravity, in this case far external to turning ankle. In reality the man is turning on his fixed astragalus. Note how a toeing out of the foot (the civilized foot) will tend to prevent the ankle from rolling over into inversion and thus saving itself. Resultant and twisting stresses are same as in preceding. Arrow 2 shows resultant of eversion. Arrow 3 shows direction of twisting stress on external malleolus due to the lever of rotation. Arrow 4 shows direction of twisting stress on internal malleolus due to the lever of rotation.

ankle outward. This tremendous compressional force must be disseminated and is only distributed at the expense of all the ankle tissues, bony and soft. This is the dynamics of compression and it is always greatest on the fibula side.

Under compression, a column or a strut is subject to leverage breaking stress also, but

with modification: a much smaller stress will serve to fracture.

Every engineer is instructed that during the test of a boiler under hydrostatic pressure he must be especially careful that the boiler be not subjected to any sudden blow. The same thing applies to an iron or steel support undergoing compression. Rupture is produced by a slight additional stress.

The astragalus cannot turn but it is closely applied to the side of the external malleolus and is applying pressure. There is clearly a stress of intensity and the tendency would be to break exactly at the fulcrum, because under compression it would require a very slight additional force and the fracture might be fragmented especially where the compression approached the elastic limit of the breaking bone because in the terminology of the mechanical engineer, bone is cold short and therefore subject to fracture from a small force acting as impact or as a transverse breaking stress.

The fracture is the result of compression plus leverage in two directions. The terminology is not clear. One man uses abduction to mean the turning outward of the whole foot and eversion to mean a rotation outward of the forward part of the foot. Another uses both terms to mean a turning outward of the whole foot and rotation to mean the twist of the forward part of the foot outward or inward.

We use the term abduction and eversion as synonymous: abduct, to withdraw from the body axis; evert, to turn out. Therefore, the entire foot is withdrawn or turned out. The term rotation means the twisting of the foot on the center of the astragalus as a pivot, therefore, the forward part only is turned out. These two stresses are simultaneous.

A pure abduction or eversion without external rotation is a rarity.

A pure adduction or inversion without internal rotation is an impossibility.

There are two distinct levers formed: first, the lever of abduction or eversion, and, second, the lever of rotation.

As can be plainly seen by glancing at Figures 4 and 5 there are two distinct mechanical entities involved in these eversion fractures. The first is produced while the center of gravity of the entire body remains internal to the foot, potentially turning outward on the astragalus. The second, while the center of gravity, is external to the foot. In the first method, the foot is the moving portion, the lever of abduction is a short lever, a pinch lever, the power applied below, the fulcrum is the inferior tibiofibular ligaments, and the weight is the weight of the man, but the strength of the lever is only the tensile strength of the inferior tibiofibular ligaments or of the external malleolus, and this is under compression almost to its elastic limit. The bending and breaking moments of a short rigid lever are the same and if the inferior tibiofibular ligaments hold, the fibula breaks at the joint level (Fig. 4).

In the second method the foot is the fixed portion, the man's weight being the power as he falls outward. The fulcrum is the point of contact of the tibia and fibula and the weight is not only the weight of the foot, but of the foot fixed under compression and, therefore, the entire weight of the man (Fig. 5).

Here is a long lever with the fulcrum a little higher up than in the other case. A long lever whose bending and breaking moments are not the same will tend to break between the fulcrum and the power, but nearer the fulcrum in exact ratio to the nearness of its breaking and bending moments, and as the fibula is comparatively rigid, it will break with frequency at the fulcrum. It would do this even more often except for the anatomical fact that it is very much weaker at a point 2.5 to 3 inches above the tip. Therefore, occasionally, especially by this second method of production, the fibula breaks high up.

But you say that all these fractures at the level of the joint, numerically greater than the classical type, are not Pott's, especially those produced by this first mechanics of ours, where the line of gravity is internal to the ankle undergoing stress.

If you will fashion two pieces of wood as the bones are shaped, or approximating them, and fasten them immovably at the points corresponding to the inferior tibiofibular articulation and apply force in the direction indicated by the first method: i.e., outwardly against the

external malleolus, you will find that you will get a fracture at the level of the joint.

If you will make the junction with a rubber band, allowing only a very little play, you will find, provided your artificial fibula is about the same strength throughout its entire length, that occasionally you will get a break higher up: not often, but occasionally. If you model your artificial fibula so that, 2.5 to 3 inches above the joint, its area is to that below, as the area of the normal fibula, that is, roughly as 1 is to 4, and allow for a little elastic motion at the artificial tibiofibular joint, you will many times get a fracture 2.5 to 3 inches above. So that the explanation seems clear. There may be occasionally enough play in these ligaments to vitiate the fulcrum at this point, or, if the stress is great, and the impact severe, the ligaments may rupture, or give enough to render useless the normal fulcrum of our lever. Instantly the fulcrum changes to a point higher up, to the lower surface of a ligamentous structure infinitely stronger than the inferior tibiofibular ligaments, the interosseous membrane, and as 2.5 to 3 inches above the tip is the weakest point of the lever and is near to the fulcrum of the new lever, it breaks at that point.

The majority of the milder types of injury are produced by the first mechanics. The majority of the more severe are produced by the latter mechanics, but, since the results are identical, and the stress the same, save in intensity, it is a distinction without a difference, and if one is a Pott's, so is the other, serious or mild.

The third group contend that these fractures are, in the main, due to rotation of the foot. If they would let it go at that there would be reason in their contention, since it is certain that next to compression this is the most important movement and not only aids in breaking the fibula but is always responsible for the broken internal malleolus in eversion fractures and for the broken external malleolus in inversion fractures. It is also responsible for the broken tibiofibular ligaments, especially when this occurs without fracture, but most of this group go further and claim that the internal malleolus is first pulled off by the strain of the internal lateral ligament, or that

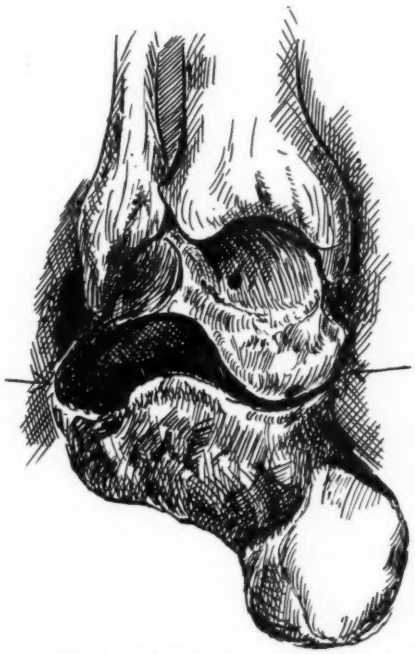

Fig. 6. This shows how in an inverting foot, there is separation at the external calcaneo-astragaloid joint. It falls short of dislocation because the internal calcaneo-astragaloid joint which is not seen in this view is at a higher level. This explains why the middle fasciculus of the external lateral ligament is so often injured in these inverting ankle injuries. Nothing of this kind can happen in the everting foot, but instead the greater the eversion the tighter these bones are locked, except for a slight lateral slip in a straight eversion.

the ligament is broken. This internal lateral ligament seems to be an ever present obsession.

We know that the distance from the astragalus forward to the toes is much greater than the distance from the same point to the posterior end of the os calcis in almost the proportion of 4 to 1, and that the foot is converted into a lever, the long arm in front, the pivot being the middle of the astragalus.

This is the lever of rotation (see Fig. 2). It may be called a lever of the first class if we choose to consider the external malleolus as the fulcrum and the point of contact on the internal malleolus as the point of application of the weight.

It could equally as well be called a lever of the second class, if we take the internal

Fig. 7.

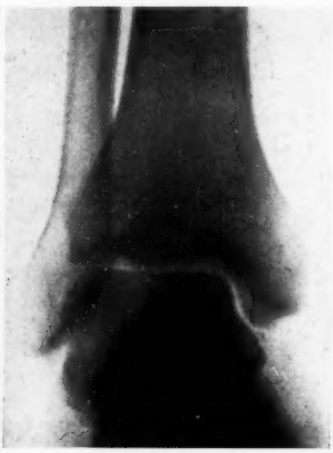

Fig. 8.

Fig. 9.

Fig. 7. Roentgenogram of an eversion injury. Practically it is a Pott's fracture without the fracture. The inferior tibiofibular ligaments are ruptured. There is, therefore, destruction of the mortise and here there is some outward dislocation. Only case of this type in our series. Very rare, 0.4 per cent of our series, but 12.5 per cent showed the feature of ruptured inferior tibiofibular ligaments in association with other types. This represents only those which showed this feature clearly in the roentgenogram plate.

Fig. 8. Type 1.—Simplest type of eversion injury. Periosteal tear of fasciculus of internal lateral ligament which runs down from the tip of the internal malleolus to the sustentaculum tali (0.8 per cent of our series). This is a much rarer injury than the periosteal tear of the external lateral ligament from inversion which is commonly due to the easier separation of the external calcaneo-astragaloid joint.

Fig. 9. Type 2.—Eversion fracture. Mild type of leverage fracture. Note large expansion of calcaneo-astragaloid joint. The cause of this injury is a stress of low intensity and the sliding over of the expansion of the os calcis at the astragaloid os calcis joint, striking the tip of the fibula. If the inferior tibiofibular ligament is not ruptured, it is of small importance and weight bearing may be permitted with care early (2.81 per cent of our series).

malleolus as the fulcrum and the external malleolus as the point of application of the weight.

This lever of rotation is a short lever, a pinch lever, but in our case the lever is stronger than either the fulcrum or the weight to be overcome, because the fulcrum is only as strong as the tensile strength of the external malleolus, and while the weight to be lifted is still the man's entire weight, yet practically this is also limited to the tensile strength of the internal malleolus.

Which one will break?

In eversion the external malleolus has already imposed upon it by other stresses and especially the compressional, a greater strain than the internal. If, for no other reason, it would break oftener than the internal but certainly one or both will break because both are weaker than the lever and if under the conditions of that particular stress their strength is equalized, they will both break, but if both break, they will break simultaneous, *because a lever is destroyed the instant that anyone of its components gives way.*

Here then we claim to have demolished the contention of the third group in so far as the internal lateral ligament is concerned, because if the astragalus is wedged against the internal malleolus from behind forward it is certain that no portion of the internal lateral ligament can be stressed or torn until one of the malleoli breaks, because between the two fixed malleoli, the astragalus cannot rotate.

If the internal malleolus breaks, the ligament is free and can suffer no harm. If the external malleolus breaks, or the inferior tibiofibular ligaments tear, the internal lateral ligament can only be injured as the result of the subsequently dislocating foot.

This disposes of all the groups and we believe we have demonstrated all of our points.

INVERSION TYPE

Why is it that there are so few of these inversion fractures in comparison to the

Fig. 10.

Fig. 11.

Fig. 10 (at left). Type 3.—Eversion fracture. Sub. Div. A. Only the internal malleolus has suffered. The internal malleolus is apt to be displaced anteriorly if due to eversion, and this is one of the diagnostic points of eversion. This half way fracture of the internal malleolus is not uncommon, due to the twist of the astragalus. Many times such a lesion occurs when the compression feature of these injuries (weight) is not present, but not always. This type A and B represent 6 per cent of our series where it was clearly eversion.

Fig. 11. Type 3.—Eversion fracture. Sub. Div. B. Same mechanics as before but more apt to result when compression is a feature. The external malleolus has resisted the stresses, but the inferior tibiofibular ligaments have ruptured, thus permitting dislocation. The internal lateral ligament is intact. Notice how impossible it would be to apply the ordinary definition of a Pott's or a Dupuytren's to this injury. Nevertheless the mechanics are the same and practically the pathology is the same. What difference would an additional broken fibula make?

eversion? It is certain that for every man who turns his foot outward, there are ten who turn it inward. There are probably many more injuries due to inversion than to eversion, but the pathology is that of an ankle strain or sprain in the great majority. A serious injury is escaped many times because if he turns his ankle inward, he involuntarily applies the principle of jiu jitsu. In other words, he gives with the force and falls, thus distributing the stress.

Try it with your foot—both in the position of inversion and eversion, the weight on the foot under strain—and see how easily you can give in the inversion position, and how impossible it is with an everting foot.

Now there is no stress in this form of injury tending to pry the external malleolus directly outward, as in the eversion type. Then why should the external malleolus break at all? Is this breaking of the external malleolus in inversion fractures due to avulsion of the malleolus by the external lateral ligament, as is claimed?

The tibia is under compression as we have shown and the astragalus cannot turn. There is no such lever of eccentricity as in the eversion type except posteriorly, but there are the static and impact compressional stresses exactly as in eversion. The break is because of the lever of rotation and often you will find the external malleolus displaced forward instead of backward, especially where there is fracture of the external malleolus alone from inversion.

Backward dislocation is a far more frequent accompaniment of this type of injury and is due to the greater stress suffered in serious cases, because of the greater latitude of movement at the various articular surfaces in this position. The very same factor which, in mild stresses enables him to save his joint from any injury, in serious stresses acts to increase the force, because even the slight increase of

Fig. 12.

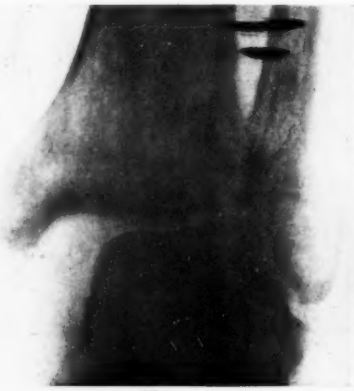

Fig. 13.

Fig. 14.

Fig. 12. Type 4.—Eversion fracture. Sub. Div. A. A leverage fracture. Break of the fibula clearly at joint level. Some displacement outward of lower fibula fragment. This is the most common type. No break of internal lateral ligament. No fracture of the internal malleolus (42.1 per cent of our series).

Fig. 13. Type 4—Eversion fracture. Sub. Div. A. Great outward displacement. The internal lateral ligament may or may not be ruptured in this type, but if so, is usually not entirely ruptured, but torn. The tearing is directly dependent on the degree of the external displacement, and is rarely tremendous, even with great displacement. The fracture of the external malleolus is clearly at the joint level, but runs up posteriorly (42.1 per cent of our series).

Fig. 14. Type 4.—Eversion. Sub. Div. B. Same mechanics, but fracture of fibula is high up. In this case only a short distance above the joint level, but often higher (4.8 per cent of our series).

latitude gives an added momentum to the stress. Figure 22 shows what a tremendous injury is possible from simply stepping from a four inch curbing with inversion of the foot.

There remains only to be considered the splits of the tibia which occur both alone, and as complications, and these may be divided into three classes, ordinarily:

1. Posterior splits of the tibia, with or without other injury, and these are extremely common (Fig. 25).

2. Anterior splits of the tibia, which are rarer than the posterior, but by no means uncommon (Fig. 27).

3. Longitudinal splits which run up the bone for some distance without being complete. These are in the nature of what is known as slips in mechanics and are comparatively rare.

All these splits of the tibia are due to impact primarily, and eccentricity of the compressional stress is many times the determining factor of the posterior split.

Classification

If we are to abandon the old nomenclature, how shall we classify these various injuries?

Simple Fractures

Simple fractures of the fibula or fractures of both bones of leg from direct violence are neither eversion or inversion fractures. They are due to direct violence or to simple cross breaking strains and are not to be considered under compression leverage fractures.

Compression Leverage Fractures or Injuries of the Ankle

Compression leverage fractures may be divided into two classes: (1) eversion, (2) inversion.

Eversion Fractures

Type 1. Simple periosteal tear of internal lateral ligament or that portion of internal lateral ligament that runs from tip of internal malleolus to sustentaculum tali of os calcis. In 249 cases it was observed only twice. This is the simplest form of eversion injury at the ankle joint and is extremely rare. It is interesting as showing that occasionally there is some separation of the internal astragalocalcaneal joint (Fig. 8).

Type 2. Fracture of the external malleolus alone half way between the joint surface and the tip. This is an eversion leverage fracture

Fig. 15.

Fig. 16.

Fig. 15 (at left). Type 5.—Eversion. Sub. Div. A. External malleolus broken clearly at the joint level. Internal malleolus also broken. No rupture of internal lateral ligament occurs in these cases. Dislocation of foot is outward and backward. Internal malleolus is broken at joint level oftenest, but a larger portion may be broken from the tibia. Mechanics as given in text. The rotation of the astragalus broke the internal malleolus simultaneously with the external malleolus (13.6 per cent of our series).

Fig. 16. Type 5.—Eversion. Sub. Div. B. Same mechanics as A, but because of undue mobility at inferior tibiofibular junction, allowing more play or because of the stresses by the second method of production, and decrease in size of fibula above, the bone breaks high up. Percentage of broken internal malleoli is greater in these high breaks, because the stresses are not disseminated as quickly as in the Type 4 cases, and are greater. External dislocation is apt not to be so pronounced, while the posterior dislocation is apt to be more pronounced. Internal lateral ligament is not broken. A classical Pott's (6.4 per cent of our series only).

due to the impingement of the external articular rim of the os calcis as the os calcis slides outward on the astragalus and the rim of the articulation brings up against the tip of the external malleolus. Such a fracture is usually due to a stress of low intensity and direct eversion, the twist being in abeyance. The external malleolus breaks half way on the transverse ligament as a fulcrum. Displacement is not usual. The mortise of the ankle joint is usually not damaged to any great extent, the inferior tibiofibular ligaments not being ruptured. Weight bearing may be begun early. Dislocation of the foot is not possible and does not take place, unless the inferior tibiofibular ligaments are ruptured, which is very rare (Fig. 9).

Type 3. Fracture of the internal malleolus from eversion (Figs. 10 and 11). There are in reality two types:

Sub. Div. A. The mildest type is usually the result of the rotation stress, the foot being in extreme dorsiflexion so that the strain falls on the portion of the internal malleolus anterior to the notch and results in a break below the joint level.

If the inferior tibiofibular ligaments are not ruptured, which is often the case, there is no dislocation possible and the type is no more serious than Type 2. This type often occurs when the compression stress is not on the foot and we have called this type "the Polo fracture" because we have had several which were produced by the opponent's horse, catching

Fig. 17.

Fig. 18.

Fig. 19.

Fig. 17. Shows the characteristic forward displacement of the internal malleolus which we claim is diagnostic of an eversion fracture when the internal malleolus is broken. Rarely is it present in the inversion type. 41.3 per cent of our series, where the internal malleolus was broken, showed this displacement, and this represents all of the cases without ruling out those showing no displacement or the ones clearly due to inversion which should be excluded.

Fig. 18. Type 1.—Inversion fracture. Middle fasciculus of external lateral ligament is injured. Periosteal insertion fracture. This is common but escapes attention many times because not X-rayed. Of little importance—5.6 per cent represent our series. But this is not the true sequence. This is probably numerically the most common of all injuries at the ankle.

Fig. 19. Inversion. Type 2.—The stress falls at base of the internal malleolus which breaks, the break many times running upward. The malleolus is displaced inward or inward and backward if at all, but unlike eversion breaks, it is rarely displaced forward. Fibula intact. Many times it is impossible to tell so that these figures represent only those clearly inversion, but it is certain that inversion breaks of this type are more common (3.2 per cent of our series).

the foot of the man and twisting it outward. Where the compression feature is not in evidence, it is possible that there is a certain amount of turning motion possible to the astragalus which permits the internal edge of the astragalus to drop: thus making these half way breaks more likely (Fig. 10).

Sub. Div. B. (See Fig. 11). The internal malleolus is broken at the joint level. The fibula is unbroken but many times the inferior tibiofibula ligaments are ruptured. If so, dislocation of the foot outward is possible and is many times present. The fragment of the internal malleolus is displaced outward by the pull of the internal lateral ligament, if dislocation of the foot occurs. Fracture of the fibula does not occur because the tibiofibula ligaments give way first and distribute the stress, or the stress is comparatively mild and is disseminated with the break of the internal malleolus.

Type 4. (Figs. 12 and 13.) Sub. Div. A. Fracture of fibula, alone at level of joint. More frequently the split runs upward, and backward, due to the combination of stresses, outward and from before backward. This is clearly a leverage fracture with the potentiality of displacement, whether or not it occurs. It is due to three stresses; eversion of the foot, the astragalus being crowded over against the external malleolus, and exerting pressure outward; a rotating force by which an additional stress is brought to bear against the external malleolus from before backward, and which accounts for the backwardly displaced lower fragment of the fibula which is observed so many times; and third, the compression stress which is present in all these cases, due to the weight of the man plus impact. The inferior tibiofibular ligaments are always injured in this type of case and often completely ruptured. Eccentricity being always on the fibula side bringing the acme of stress to this side, accounts for the preponderance of fibula fractures.

In our series of 249 cases, 55.4 per cent were fractures of the fibula alone, and 42.1 per cent belong to this type of our classification.

Fig. 20.

Fig. 21.

Fig. 20 (at left). Type 2.—Inversion. Same as Figure 19, but with displacement and dislocation in a young subject.

Fig. 21. Type 3.—Inversion. A very rare type, the tendency being to break the internal malleolus and thus free the pressure, or to break both bones simultaneously. If, however, as occasionally happens, the internal malleolus resists the strain, the external malleolus may be broken alone either at the joint or above as in this case. This is much more apt to happen if the foot is in extreme plantar flexion, because, in dorsiflexion, there is the tendency to anterior dislocation and the points of contact are more apt to be relieved from pressure, especially if anterior dislocation occurs. In plantar flexion the posterior outer edge of the astragalus is raised so that the contact with the external malleolus is greater and a rotating stress is more apt to catch it, the pressure being from behind forward. The existence of this type has been denied, nevertheless occasionally it occurs. Rupture of the inferior tibiofibular ligaments sometimes saves the fibula from fracture.

Type 4. Sub. Div. B. (Fig. 14.) The fibula broken alone but higher up than the joint level. Same mechanics as A, and all other conclusions pertaining to A apply to B. The high fracture of the fibula is explained in this article, and is more likely to occur in that form of eversion injury where the center of gravity is outside the stressed foot. Only 4.8 per cent of our series fall in this group.

Type 5. Fracture of fibula and internal malleolus. Sub. Div. A. (Fig. 15.) The fracture of the fibula being at the joint level. The same mechanics as Type 4 and the same conclusions apply. The fractured internal malleolus is a matter of small importance unless so badly displaced as to resist replacement. The internal malleolus is many times displaced somewhat forward, a feature which we contend is always a proof of the eversion character of the injury (Fig 17). Dislocation, as in Type 4, is a potentiality, whether or not it occurs. The internal malleolus is nearly always broken at joint level; 13.6 per cent.

Type 5. Sub. Div. B. (Fig. 16.) Fracture of the fibula high up and fracture of internal malleolus at or near level of joint as usual. The same mechanics as A of Type 5 and as Type 4, and same conclusions, apply; 6.4 per cent fall into this class. This is the classical Pott's.

Inversion Fractures, 13.6 per cent

Type 1. (Fig. 18). Insertion fracture, periosteal tear of the external lateral ligament. A small chip of bone is separated from the tip of the fibula by the stress suffered by the middle fasciculus of the external lateral ligament. This is a most common type of injury and is caused by the easy separation of the external calcaneo-astragaloid joint, when the foot is inverted. In our series, there were 5.6 per cent but it is probable that this form of injury represents the largest type of injuries to the ankle, since it is probable that few of these cases are X-rayed. The injury is simple, and a few days of restriction is all that is usually required with subsequent care.

Fig. 22.

Fig. 23.

Fig. 24.

Fig. 22. Type 4, A. Inversion. Inversion fracture of fibula and internal malleolus. Often accompanied as here with impact complications. The fracture of the fibula is at the joint level and the internal malleolus at the joint level but often includes a large piece integral with the malleolus. Many times dislocation internal and posterior. It is obvious that a recovery here is only possible by operation and even then doubtful. Quick motion here is out of the question.

Fig. 23. Type 4, B. Inversion. The fibula is broken high up. The large piece integral with internal malleolus shows. Internal dislocation is reduced, but there is still backward dislocation. Reduced, this fracture will respond almost as quickly as one apparently much less serious. (Sub. Div. A and B represent 3.6 per cent.)

Fig. 24. Central split of tibia breaking out both anteriorly and posteriorly with telescoping. This is an operative case and result is extremely doubtful. To apply motion quickly to such a case, even if it could be reduced without operation, or to apply any fixed rule would be, of course, absurd.

Type 2. (Figs. 19 and 20.) Inversion fracture of the internal malleolus. This is a common type of inversion fracture. The stresses are not distributed, the strain comes against the base of the internal malleolus; the same three stresses which we have mentioned in the production of the eversion types but in this case reversed. No such lever of eccentricity is present on this side, however, except posteriorly, sometimes, and, therefore, the fracture of the internal malleolus is a sheer fracture, usually; the compression, however, preventing the astragalus from turning. The fracture begins at the joint surface and either runs transversely or up to include a somewhat larger piece. Compressional splintering is not present on this side.

Dislocation of the foot is a potentiality, whether or not it occurs. The fragment is displaced inward, if displaced at all, and sometimes backward, but, unlike the eversion type, is not likely to be displaced forward. This represents a differentiation, where the history is not clear.

Rupture of the inferior tibiofibular ligaments may occur, but is less likely than in the eversion types of injury. Of this type 3.2 per cent represent our series, but this is clearly erroneous, because many times where displacement or dislocation does not occur, the history will not be clear. Many more of these cases were caused by inversion than are shown by these figures, but fracture of the internal malleolus, alone, is, nevertheless, more often the result of eversion than of inversion.

Type 3. (Fig. 21.) Fracture of the fibula from inversion either at the joint or high up. This is rare, but happens occasionally. The inferior tibiofibular ligaments are usually injured, but dislocation rarely occurs.

Type 4. Fracture of fibula and internal malleolus. 3.6 per cent clearly inversion.

Sub. Div. A. (Fig. 22.) Broken fibula and internal malleolus. Fibula at joint level. The

Fig. 25.

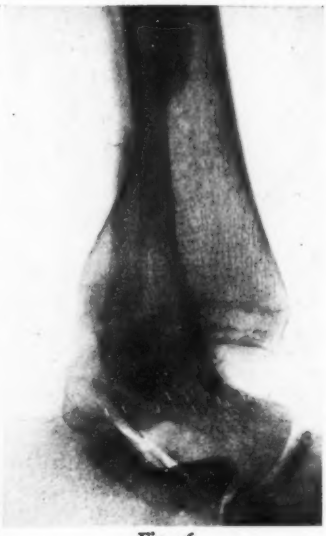

Fig. 26.

Fig. 25 (at left). Impact splits of tibia. Posterior split with fragment forced upward. No dislocation of the ankle. These may occur alone or as complication of any of the other types. This is the result of impact in plantar flexion.

Fig. 26. Impact split of tibia. Posterior with great posterior dislocation. Anterior and posterior 1.0 per cent of ankle fractures alone; 11.2 per cent as complication of other fractures. Longitudinal splits of the tibia are occasionally encountered.

same mechanics reversed as for the same type of eversion fracture. If displacement of the fragments occurs, the internal malleolus is not displaced forward, as in the eversion type, and, unless the foot dislocates, the lower fragment of the fibula is not so likely to be displaced backward. Dislocation is common, however, changing the picture.

Type 4. Sub. Div. B. (Fig. 23.) Fracture of fibula high up and fracture of internal malleolus. In our series this was less frequent then the Sub. Div. A, in the proportion of 1 to 2. Exactly the same mechanics apply here, and the same conclusion may be drawn. Dislocation is common, and backward dislocation is a more common accompaniment of inversion fractures than of eversion.

There is another type of injury to the ankle joint usually the result of rotation, which without a solution of continuity of any osseus structure, presents much the same condition, inasmuch as the inferior tibiofibular ligaments are ruptured, the mortise of the ankle joint is broken up, and dislocation is not only a potentiality, but is frequently present. Such a case is represented by Figure 7, and shows clearly the outward dislocation resulting from a broken inferior tibiofibular ligament. Inasmuch as soft tissue tends to heal more quickly than bony tissue, no such period of immobilization is necessary, but restriction of motion and weight bearing for a time is absolutely essential to the restoration of the efficiency of the astragalotibiofibular mortise.

TREATMENT

Reduction where there is dislocation is the important point and is usually easy of accomplishment. Even in the simple cases of fractured fibula from eversion either low down or high up, the same force which produces the break is apt to displace posteriorly the lower fibula fragment and to displace the astragalus backward a little, which is often missed in the study of the X-ray plate, and disregarded in

Fig. 27.

Fig. 28.

Fig. 27 (at left). Impact splits of tibia, anterior split. Due to impact with foot in dorsiflexion. Less frequent than the posterior splits.

Fig. 28. Meissonneuve's fracture, an eversion fracture, very rare. Fracture of the internal malleolus. Fracture of the fibula high up. Note fracture of fibula shows tortional stress. This is a compression fracture presenting no fundamental differences from the classical type as the height of the fibula fracture is not a matter of importance. Outward dislocation of foot.

treatment. First, flex the knee. Rocking the foot usually suffices to correct the lateral displacement, but many of these minor posterior dislocations are not reduced. Direct pressure backward on the intact tibia above, and a strong pull forward on the tuberosity of the os calcis carrying the foot into hyperdorsal flexion. By so doing, we shall save the patient much subsequent discomfort. With direct pressure, we can also correct these backwardly carried inferior fragments of the fibula, and this is essential. Twist the foot inward and forward. It twisted outward and backward when it occurred. After reduction, there is very little tendency to displacement, unless a posterior split has occurred. If the internal malleolus is intact adduction is the position of choice, and may be held by any splint. Personally for the emergency we use a pillow splint tied with tapes and reinforced with pieces of splint wood or board, internal, external, and posterior. Below the foot, the open ends of the pillow case are folded and pinned, so as to keep the foot hyperdorsiflexed. If the position is satisfactory in the X-ray, we put on a Cabot splint, bending the knee always to keep the position of hyperdorsiflexion of the foot. This relaxes the gastrocnemius and solius muscles, which form the tendo achillis. A doughnut is placed under the heel always. No Pott's should be put into plaster at this time. The swelling is too great and the cast is always inefficient under these conditions. If the internal malleolus is broken, we do not invert the foot. We use a straight position. Inversion with a broken internal malleolus is an absurdity; worse than an absurdity, it is a menace.

Where the internal malleolus is broken and displaced, Magnuson of Chicago uses a small incision over the malleolus and pins it back into place with an ivory pin which he leaves. He believes that by this manœuvre, he restores the mortise of the joint, and that then he can place the foot in the position of inversion. We believe the reduction and the straight position are sufficient in most cases, but in a very limited number, where reposition of fragments is hard to obtain, this treatment is ideal.

We always have several pillows under the knee to keep it in the flexed position, thus rendering the hyperdorsiflexed foot comfortable. Only in those cases showing a large anterior split of the tibia which are rare, do we vary the hyperdorsiflexion of the foot. Even in these cases, we are careful to get the foot above a right angle, because we have found that this is one of the movements easily lost by disuse and slow in recovery. Keep the foot above a right angle with the leg, and you will have no difficulty, even where there is a tendency to new bone on the anterior edge of the tibia. If there is a posterior split of the tibia, it is essential to keep the foot dorsally flexed; a plantar flexed foot is apt not only to redislocate where the fragment is large and involves at least half the articular surface of the tibia; but it is apt to push upward the posterior fragment.

Fortunately, these posterior splits are usually small and do not involve a great articular surface.

We begin motion, in most of these cases, by the third or fourth day in the ordinary type, and certainly by the end of the first week in nearly all of them, unless there is tremendous swelling or tremendous tearing of ligaments which only happens in the very serious cases.

We believe that if one observes carefully, he will be surprised at how rarely this occurs. Every day, thereafter, the foot is gently moved passively and actively, the hyperflexed foot being carefully lifted from its splint by the toes, the knee kept well flexed. We follow the same manœuvre in all our cases whether the fibula is broken high up or low down. The broken internal malleolus simply means a greater degree of caution. Plantar flexion must never be carried to extremes. An arc of 20 degrees of motion at first is sufficient.

Dorsal flexion, actively and strongly, is essential, and effort is made by the patient early to dorsiflex his foot strongly, except in cases of anterior split. Even here, passive flexion within limits is used from the first.

In the case of posterior splits, be especially careful not to plantar flex too quickly or too far. In some few cases of great injury, we permit immobilization for 10 to 15 days with the toes free for movement, but in most cases we begin motion gently, not later than the fourth day. Thereafter, motion every day, and as passive is to be preferred to immobilization, so active motion, no matter how limited, is to be preferred to passive motion. Motion must be retained from the first, and can be so retained in practically every case, if care and intelligence are used in the manipulation. This is the point of the whole treatment. Restriction of motion—partial ankylosis—is due to organization of inflammatory products, and involvement of tendons, and tendon sheaths in the reparative scar tissue, together with a traumatic synovitis which is kept without motion. It is remarkable how quickly these immobilized ankles tend to stiffen. It is remarkable how quickly and easily and without pain they take up their functions, even in the face of tremendous trauma, if they are given a chance early. Do not let them stiffen. If you do, you will have impaired function, for a long time.

By the end of the first week, or before, in nearly all of these cases, the swelling will be of such a nature that a cast is practicable. It should extend in the simple cases only a short distance up the leg. In the more serious, it should reach nearly to the knee.

If the internal malleolus is unbroken, the assistant holds the foot by the toes in strong inversion and hyperdorsiflexion with a flexed knee, while we are molding the cast. Get well above a right angle, and use a light cast.

The cast is put on, over stockinette, and the stockinette is not pulled over the foot. It is rolled over, thus preventing any undue pressure on the broken malleoli. A few small strips of metal are incorporated under the heel to prevent subsequent breaking and the cast is cut down the front, when sufficiently hard, a strip 1 inch in width being removed along the whole length and widened from the ankle forward to from 1.5 to 2 inches, so as to permit of easy removal. Many of these cases have a molded leather ankle splint by the end of the first week because of its lightness.

After the tenth to the twelfth day, all danger of damage to these injured ankles has passed, if we use care and all of them are in plaster or molded leather, the patient going about on crutches. After the first few days the simple cases are soaked in very hot water once a day, and carried through their exercises. We use the same treatment in these cases as we have advocated in Colles fractures for many years, but with one difference. We have repeatedly made the statement that an ordinary Colles fracture with anything for retention except a wrist strap after the tenth to fourteenth day was maltreated. We do not mean by that, that a patient who has suffered a Colles fracture can throw a base ball on the fourteenth day, or the twenty-first, but we do say that there being no weight bearing at the wrist, we may dispense with all restrictive apparatus, except the small leather wrist strap, very early. The same would apply to a Pott's except for three factors: the weight bearing; the weight of the foot itself anterior to the astragalus; and the pull of the gastrocnemius and solius muscles. These, then, must be considered. After the twelfth day, there is little danger of redislocation or damage to the broken parts provided we have no weight thrown upon the foot. We cannot permit weight bearing in any of these injuries at the ankle joint due to leverage until there is no possibility that that weight bearing will cause deformity by bending out the bones on the hinge of soft callus, which holds them together, and we must not permit this until such time

as this soft callus has been replaced by bony tissue strong enough to resist the pressure which it will be called upon to bear.

This is an individual proposition and probably differs greatly in every person, so that we must not take chances. One bad result—one foot that turns out progressively after weight bearing—will more than counterbalance good results in a dozen cases. It is probable, in fact I am convinced, that many of these cases would receive no injury from weight bearing much earlier than we now permit, but, where the individual coefficient of repair is an unknown quantity, we must not take chances, and there is no necessity for so doing. With motion each day, by the twenty-first day, all these cases will be able to have a practically normal motion at the ankle, the simple ones much sooner. The ankle will swell and in some of them cyanosis will be pronounced, but they will all have the normal range with perhaps some slight restriction. So long as we preserve this normal motion against the day when we can, with absolute safety, permit of weight bearing, we shall have done all that is needed to do, since in this case they will be able to walk with ease when the time comes, instead of having the recovery of lost motion still before them.

In the simple case of a break of the external malleolus below the joint level, there is no tendency to eversion of the foot. Such a case needs practically no restriction, except care, and may bear some weight with care at almost any time, certainly by the twelfth or fourteenth day. A case of simple break of the fibula above the joint by direct violence, and not a leverage fracture, is held together by soft callus by the twelfth day. There is beginning actual bony growth across the break by the twenty-first day. There is little strain in weight bearing in such a case, and neither does it require a great amount of restriction. By the twentieth day, such an injury may safely begin to bear some weight, but care must be observed.

In a leverage fracture of the ankle joint, attended by ligamentous tear, and by the potentiality, at least of dislocation at the astragaloid joint, either by inversion or eversion, with fracture high up or low down of the fibula, by the fifteenth day, the foot should be out of the cast for an hour or two each day. It should be soaked in hot water, and active motion in flexion and extension should, by this time, reach a fairly normal range. No lateral motions should be permitted.

By the eighteenth day, only a light molded leather ankle strap should be used by day, the cast being used only at night, but the patient should be permitted to sit with the foot resting lightly on the floor, without weight and in the simple cases with a shoe raised ¼ to ⅓ inch on the inner side, slight weight bearing may be begun by the twenty-first to thirtieth day, but no great amount of weight should be borne for, at least, 4 or 5 weeks. We must remember that with this treatment when he begins to bear weight, the surgeon has finished the case, and we can therefore afford to go slow. There are no long months of struggle before him for the completion of motion.

In the serious cases it is better to wait 6 weeks before weight bearing. It is safer to do so, and there is little to be gained, since the motion has not been lost. In the serious cases with great injury and dislocation, where the man is very heavy, a steel support attached to a padded ring on the leg and inserted into the heel of the shoe may be used for several weeks after walking is permitted, but in the vast majority of cases, this will not be found necessary. The inverted foot and the ankle strap will be sufficient. A leather or a metal foot plate is sometimes added.

The inferior calcaneoscaphoid ligament, the spring ligament which is one of the important ligaments of the internal longitudinal arch, is rarely injured, but all muscles and ligaments are weak from disuse after a fracture of this kind. It is, sometimes, important to wear a foot plate, but it is of far greater importance to put the injured foot through a period of regular exercise, before weight bearing is permitted. This is the treatment which should be used in every case of fracture of the ankle, and, if used, will shorten the time of disability by months. It is unfortunate that in the unintelligent class of people, care will have to be used, and the casts left on for a longer period, because they cannot be trusted, and damage suits are, at present, far too common to be

249 ANKLE INJURIES—ALL KINDS EXACTLY AS THEY CAME

I

	Cases	Per cent
Fibula alone	138	55.4
Broken high	12	4.8
Half way	7	2.8
Clearly at joint or near it	105	42.2
Periosteal tears from inversion	14	5.6
Ankle dislocated at joint	29	11.6

11.6 of whole or 21.01 per cent of number with fibula broken.

II

Fibula and internal malleolus	52	20.9
Fibula broken:		
High	16	6.4
Half way	1	.4
At joint or near	34	13.6
Periosteal tear	1	.4
Internal malleolus broken:		
At joint level	45	18.
Low	7	2.81
Dislocation of ankle	34	13.6 of whole series or 65.4 per cent of this class.
Internal malleolus displaced anteriorly	23	9.2

III

Internal malleolus alone	35	14.
At the joint level	26	10.4
Below half	7	2.81
Periosteal tear	2	.8
Dislocation of foot	12	4.8 of whole or 34.3 per cent of this type.
Internal malleolus displaced forward indicating eversion	13	5.2

IV

Fractured fibula and separated tibial epiphysis	5	2.
Dislocated foot	4	.....

V

Fractured scaphoid, alone	2	.8
As complication	2	.....

VI

Fractured astragalus, alone	5	2.
As complication	2	.....

VII

Fractured os calcis	5	2.

VIII

Dislocated foot without fracture	3	1.2
Forward	2	.8
Outward with ligament rupture	1	.4

IX

Impact splits of tibia, alone	4	1.6
As complication	28	11.2

X

Total dislocation of the ankle, all cases	87	34.9

XI

	Cases	Per cent
Clearly inversion fractures	34	13.6
Periosteal tears of fibula	14	5.6
Periosteal tear, fibula and broken internal malleolus as well	11	4.4
Broken internal malleolus, alone	8	3.2
Broken astragalus from impact in inversion	2	.8
Broken fibula and internal malleolus	9	3.6
Fibula high	3	
Fibula at joint	6	

Ruptured inferior tibiofibular ligaments clearly as evidenced by wide separation of the bones in the X-ray plate, 30 cases, 12.05 per cent.

Many others undoubtedly were ruptured but when the X-ray evidence was not conclusive they were excluded.

disregarded, but even in this case, where the casts (for reason of prudence) must be left for a much longer time than necessary, it is incumbent upon the surgeon to see that the mobility of the foot is preserved from the first.

In the eversion type, we always use a shoe which is made so that the inner side of the heel and sole make a straight line, as all shoes should be made, and long enough to sweep wide of the toes.

The inner side of the heel and sole are raised ¼ to ⅓ inch in order to invert the foot slightly. An inverted foot is always a strong foot, an everted one is always a weak one.

CONCLUSIONS

Leverage fractures of the ankle under compression are, in reality, all of the same etiology, and vary simply from distribution of force and intensity of stress. This applies to most of the so-called simple fractures of the lower end of the fibula since the salient features of this fracture are present: i.e., ligamentous and tendon injury and a destroyed mortise permitting dislocation. Simple fractures of the fibula at any level, the result of direct violence without ligamentous rupture, do not belong to this type, but there are no compression leverage fractures of this kind involving the ankle joint.

The inversion types are not usually attended with so great a destruction of ligaments, but there is an equal or more serious involvement of tendon sheaths, and a greater posterior dislocation is common in our experience.

The majority of these fractured ankles are not tremendously serious injuries and do not deserve the bad repute which is theirs at

present. The bad results have been the result of immobilization and misdirection of effort on the part of the surgeon.

Motion, active and passive, from the very beginning is the solution of the problem. Do not permit the ankle joint to stiffen in the first place. This is easy to prevent. It is only overcome, once it has occurred, at the expense of tremendous effort and months of time.

It is easy to reduce an ankle fracture, put it in a cast and look at it once a week, but it is fatal to the patient. If time is too important for one to devote 20 minutes a day to his ankle fractures, he had better turn them over to some other surgeon. No masseur is competent to do this work unless he is a surgeon and also competent to remedy any bad result. In the hands of a man who understands the pathology of the lesion, there is no danger.

From both the standpoint of economics and compensation insurance, this is a tremendously important subject. The time lost now, as a result of this injury, is enormous. It often extends over periods of months, or even a year or more, in individual cases, and this applies unfortunately to the mild type as well as the severe. Multiply this by the number of ankle fractures in a single year, and the time lost will be found to be enormous. A fracture of this type, without complications, should be returned to light employment in from 4 to 7 weeks, with a practically normal foot, except for some swelling which in certain cases will persist for a long time.

LYMPHATICOSTOMY IN INTESTINAL OBSTRUCTION[1]

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Department of Surgery, University of Toronto

THE experiments upon which I formulated the operation of lymphaticostomy in peritonitis verged on the subject of intestinal obstruction in that the appendix and mesoappendix were ligated to initiate the peritonitis, and that is comparable to the production of a small closed segmental obstruction with occluded blood supply and with continuity of the digestive tract. Because of that fact it occurred to me that the same striking results could be obtained in intestinal obstruction by draining the thoracic duct, and I undertook a series of experiments on dogs with the intention of finding the path of absorption of the toxin and how the toxin is formed.

Reference to the classical descriptions of intestinal obstruction gives a clue to the possible cause of the toxæmia. We find that the same symptoms are produced by gangrene of the bowel in thrombosis or embolism as in an actual obstruction. Acute necrosis of the pancreas is hard to distinguish from obstruction. In thrombosis and acute necrosis of the pancreas the underlying cause is death of tissue and naturally we think of the possibility of a similar cause in actual obstruction.

In dogs under ether and with aseptic precautions a series of closed segments, closed loops, and total occlusion of the duodenum and small intestine, with and without drainage of the thoracic duct, was carried out (Figs. 1, 2, and 3).

Obstruction which occurs suddenly during the activity of digestion is a more serious condition than that which occurs when the bowel is at rest. The reason for this is that during digestion there is a profuse pouring out of digestive fluids which not only causes dehydration but robs the blood of necessary ingredients which are not reabsorbed. In a dog obstructed during active digestion of food in the stomach it was estimated that from the duodenum alone approximately 2 per cent of the body weight in fluids was secreted in an hour. When one adds to this the secretions from the stomach, the liver, and the pancreas it is not difficult to understand the gravity of such a loss. But this is aside from toxæmia and this subject will be dealt with under two heads: (1) the formation of the toxin; and (2) The path of absorption of the toxin.

THE FORMATION OF THE TOXIN

In all the fatal cases there was extensive necrosis and denudation of the mucous membrane. In the closed segments and loops the necrosis usually went on to perforation, while in total occlusion the greatest destruction of tissue was at the lower end which, in some instances, progressed to gangrenous spots not perforated at death. Two factors were thought to account for these changes:

[1] Read before the Academy of Medicine in Toronto, October 16, 1923.

Fig. 2.

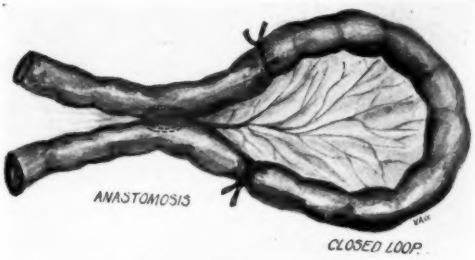
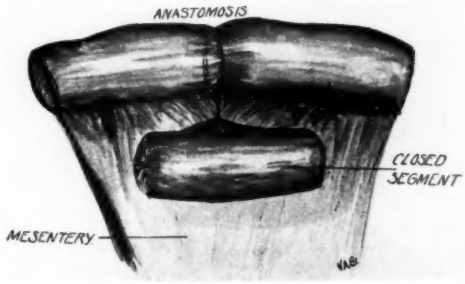


Fig. 1.



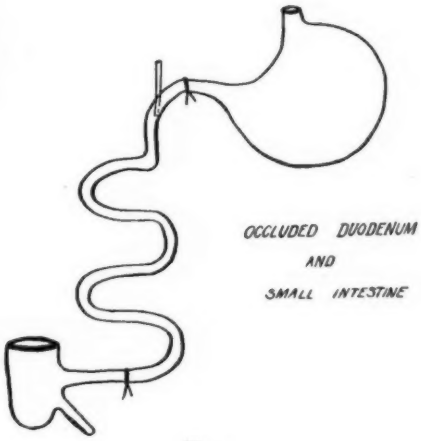


Fig. 3.

Fig. 1, 2, and 3. The varieties of obstruction.

a. Distention. This was produced by active secretion or by venous stasis from interference with the blood supply. The stasis came into play repeatedly in the closed segment obstructions where the segment was suspended by a long mesentery.

b. The action of retained digestive juices. In 34 cases of the various obstructions the average time before death in closed segments in the lower ileum was 42 hours. In closed duodenal segments death followed in 48 hours. That death was more rapid in the ileal segments than in the duodenal may be explained by the longer mesentery in which venous stasis could more easily occur, and also the addition of bacterial infection invading the peritoneal cavity. In one closed loop in the duodenum the time was 72 hours, and in one closed loop in the lower ileum 144 hours. In the closed loop group only two dogs died and eight recovered. In these cases the obstructed area was held by the bowel preventing any interference with the blood supply. The average time in total occlusion was 72 hours. When the most fatal obstruction was a closed ileal segment and one of the least fatal a total occlusion involving the entire duodenum and most of the small intestine, one could hardly say that the lethal factor was a toxin formed in the mucous membrane which is brought into activity by the obstruction in the form of a secretion, and the only explanation seem to be that the toxin was formed by the breaking down of necrosed tissue. In one case of total occlusion the upper ligation was below the pancreatic ducts. This dog died in about 40 hours and at postmortem the liver presented a most striking appearance. It was small and covered with small white necrotic areas. There was some necrosis of the pancreas. The blood was observed to look more like bile than blood. Evidently there was a backing up of the secretions which accounted for the extensive necrosis. Figure 4 shows the necrosis of the liver. This section seems to rule out the possibility of any absorption of toxins through the blood stream for if that were the case the necrosis would have commenced in the portal branches around Glisson's capsule.

Figures 5 and 6 show the mucous membrane of obstructed areas in dogs which recovered. There is an absence of necrosis. Figures 7, 8, 9, and 10 are from dogs in which the obstructions were fatal. Here extensive necrosis is seen. It is due to a cause acting from the lumen of the intestine. This destruction of tissue means that there are innumerable lacteals and lymphatics opened up making a very direct path for the absorption of any toxins within the lumen of the bowel. Figure 11 is from a dog which died of intussusception developed after it had recovered from an experimental obstruction. Here again the necrosis of the mucous membrane is very evident.

THE PATH OF ABSORPTION OF THE TOXIN

That death in thrombosis or embolism of the mesenteric vessels is due to the absorption from the gangrenous tissue can hardly be disputed, but how can absorption occur through obstructed blood vessels? The absorption is through the lymphatics, the third circulation, which makes gangrenous areas so fatal when occurring in tissue richly supplied by them. In one dog a 6 inch segment of the lower ileum was tied off with tapes and the blood supply ligated in the mesentery. In 24 hours the dog was dead, and the segment was found black, about as thin as paper and shrivelled up to about one-quarter normal size. It was lying free and not adherent to any other structure. The fluid in that gangrenous area could have been absorbed only by the lymphatics which had been left intact. An opposite picture was seen in another dog in which an area was obstructed and not only the blood supply but also the lymphatics were occluded. In 24 hours this dog was alive, not toxic, and the obstructed area was about four times normal size and filled with bloody fluid. Until it ruptured this obstruction was not fatal because the lymphatics were blocked. When the blood supply to a part is cut off but the lymphatics left intact there is still a suction on the lymphatics and any fluid present will be absorbed by them.

There were many little features of the experimentation which have thoroughly shaken the theories of absorption from the intestine and peritoneal cavity and if I may digress for a moment one instance may be of interest. During an operation the thoracic duct had been opened in the neck and there was a moderate flow of lymph which rapidly coagulated. The abdomen was opened for the production of an obstruction. A duodenal tube was passed into the lower part of the duodenum and a quantity of tap-water passed through the tube. There was almost an immediate rapid increase in the flow of lymph until it was about three times the former flow. I had never seen such a copious flow and the lymph became thinner so that it failed to coagulate. On a later occasion a similar procedure was carried out and the carotid artery was attached to a kymograph. When water was introduced into the duodenum there was again a rapid increase in the lymph flow without any alteration in the blood pressure. In another dog the abdomen had been opened and a ligation of the appendix done. The abdomen was closed and when, immediately later, the thoracic duct was exposed and opened it drained bloody chyle very freely during the whole of the operation. The blood was absent from the duct drainage several hours later and it was assumed that some oozing of blood had occurred into the peritoneal cavity when the abdomen had been opened and it had been absorbed directly into the lymphatics. These findings are evidence of a free communication between the lymphatics and the lumen of the bowel in the one instance, and the lymphatics and the peritoneal cavity in the other.

Working on the theory that toxic absorption takes place through the lymphatics, the thoracic duct was drained in the various forms of obstruction. Brief summaries of several of the experiments show the striking results obtained.

Dog 3. Ether anæsthetic. A 4 inch closed segment of the lower ileum was produced and an end-to-end anastomosis was done between the divided ends of the ileum. The thoracic duct was then drained. On the third day there was evidence of a diffuse peritonitis but the thoracic duct fistula drained successfully and these symptoms subsided. On the seventh day the dog was rapidly coming to normal. The abdomen was again opened under ether and a subsiding peritonitis found. The segment had ruptured in two places. It was removed and the dog recovered.

Dog 4. The same procedure was carried out. The duct failed to drain and the dog died in 48 hours. There was a beginning diffuse peritonitis, and the segment had ruptured as in Dog 3.

Dog 33. The same obstruction was produced but the duct was not drained. The dog died in about 48 hours. There was a diffuse peritonitis and the segment had ruptured as in the other dogs.

Dog 17. Obstructed just beyond pylorus and about 6 inches above the cæcum with double strands of chromic catgut. Drained the thoracic duct.

Dog 18. On the same day obstructed as in Dog 17 but did not drain the thoracic duct.

On the fourth day the dogs were again anæsthetised and the abdomen opened. In Dog 17 the obstructions were still complete. The intestine looked healthy and was practically empty. The obstructions were relieved and the abdomen closed. In Dog 18 the obstructions were complete. The intestine was filled with fluid contents, deeply congested throughout and at the lower end markedly distended and verging on gangrene in a number of areas. The obstructions were relieved and the abdomen closed. Dog 17 made an uneventful recovery and Dog 18 died the same day.

From the experiments the following conclusions were drawn:

1. Toxæmia in acute intestinal obstruction is due to the absorption of toxins formed from necrotic tissue.

2. When in total occlusion the upper obstruction is just below the pancreatic ducts there is rapid necrosis of tissue above the obstruction and death, but when the obstruction occurs above the ducts just beyond the pylorus even though more area is obstructed the accessory gland digestive juices, and especially that from the pancreas, are neutralized lower down and necrosis and death are considerably delayed.

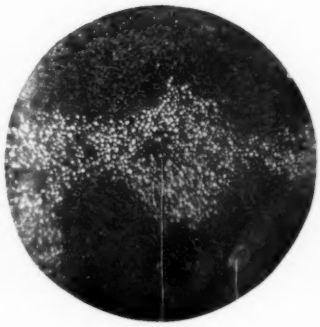


Fig. 4.

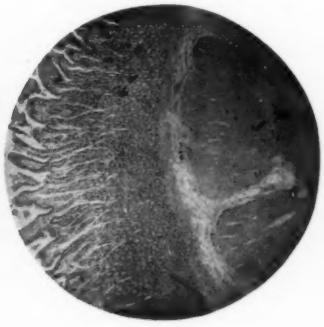


Fig. 5.

Fig. 6.

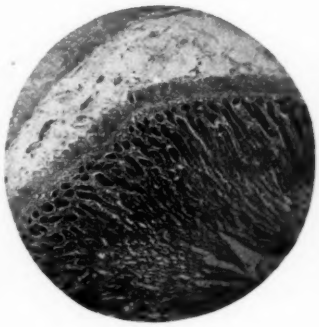


Fig. 7.

Fig. 8.

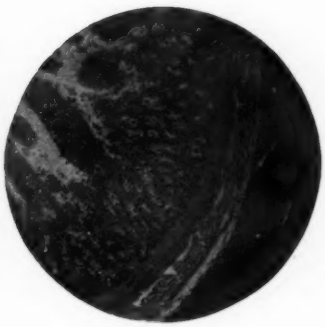


Fig. 9.

Fig. 4. Shows necrosis of the liver.
Figs. 5 and 6. Sections showing the mucous membrane of obstructed areas in dogs which recovered.
Figs. 7, 8, 9, and 10. Sections from dogs in which obstructions were fatal.
Fig. 11. Section from a dog which died of intussusception developed after it had recovered from an experimental obstruction. The necrosis of the mucous membrane is very evident.

Fig. 10.

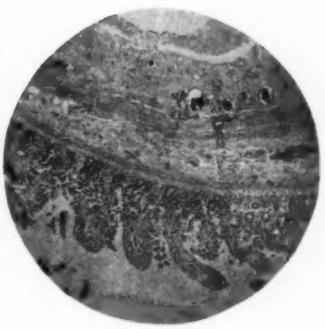


Fig. 11.

3. The absorption of toxins is through the lymphatics to the thoracic duct.

The practical application of these findings to surgery has not been worked out. Merely touching on their bearing on surgery one may say that they call for the withholding of fluids by mouth, dehydration being combated by intravenous infusions. They indicate that in all operative procedures the lymphatics should be taken into account, and to prevent a continuation of the absorption of toxins in some obstructions it may be necessary to supplement the relief of the obstruction by a lymphaticostomy. Should this be done the continual administration of water through a duodenal tube will help to keep a constant drainage from the duct and prevent blocking.

LYMPHATICOSTOMY IN PUERPERAL INFECTION

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FROM time immemorial medicine has been battling with the condition or conditions known as puerperal infection. Giant strides have been made especially since the time of Oliver Wendell Holmes and Semmelweiss who proved beyond doubt the infectious origin of the disease. Their studies and conclusions undoubtedly lessened the percentage occurring in hospital practice but in general work the condition remains about the same as it has for centuries. V. Bonney (1) states: "Sepsis accounts for between 30 to 35 per cent of total deaths during the puerperium."

Doederlein (3) classifies puerperal infection into the following:

1. Ascending type—through natural channels;
2. Descending type—through blood stream and by contiguity.

The ascending type is by far the more frequent, begins by wound infections and may be due to the following organisms named according to Williams (7) in the order of their frequency (1) streptococcus, (2) staphylococcus, (3) gonococcus, (4) bacillus coli communis, (5) bacillus aerogenes capulatis, (6) pneumococcus.

While in the milder types the infection remains localized in the genitalia or generative organs, a large percentage become systemic in character due to absorption through the blood stream and lymphatic channels. Those which localize and are treated surgically recover in nearly all instances. Those which become systemic show a mortality rate between 28 to 40 per cent, death usually being caused by exhaustion and toxæmia of peritonitis.

It has been proved that probably the majority of systemic puerperal infections originate from lymphatic absorption. Edgar (4) states: "The genesis of peritonitis in the puerperium is the lymphatics—the bacteria passing from the lymph spaces of the uterus directly into the peritoneal cavity." Southard and Canavan (5) state: "The pelvis probably surpasses the intestine in supplying regional lymph nodes with bacteria." They found in 15 out of 20 cases postmortem with pelvic lesions micro-organisms in the pelvic lymph nodes.

The treatment of these systemic infections has been mainly medical with the evolution of a "hands-off" policy. Doederlein states: "Abdominal operations in the ascending type without ocalization are out of the question." Watkins (6) states: "As the disease is chiefly systemic, treatment is essentially general. The important part of the treatment of puerperal infection is the use of remedies to increase the body resistance and the abandonment of measures that interfere with the development of immunizing substances." Costain (2) in a recent article states: "Absorption from the peritoneal cavity takes place largely through the lymphatic system and one of the most helpful procedures in relieving the toxæmia is through drainage of the left thoracic duct."

In support of his contention, I wish to present the following case:

Mrs. M. K., age 17, entered St. Mary's-Ringling Hospital, May 10, 1923, with the following history: The father, age 41, is alive and well. The mother, 40, is alive and well. She has 3 brothers and 2 sisters, alive and well. Patient had measles and chicken pox when a child, both followed by full and uneventful recovery. Menstruation began at 11 years, has always been regular, and free from pain until cessation at her first pregnancy.

Present illness. Delivered April 14, 1923—in labor for hours but delivered without instrumentation—second degree tear of perineal body and some lacerations of cervix which were not repaired. She felt fine first 48 hours, then had a chill followed by fever, when they called another physician who examined her April 18, 1923, with the following findings: Diminished lochia, lacerations of cervix and perineum, slight amount foul smelling vaginal discharge, lobar pneumonia of right base. Temperature declined by lysis and she was supposed to be free from fever from May 4 to 8, although husband states she didn't feel at all well during this time. On May 8 she took suddenly sick during the night with chill—crampy pains across lower abdomen which were accompanied by nausea and vomiting. On May 9 when seen by her physician, she had a temperature of 104—generalized tenderness over entire lower half of abdomen, and profuse vaginal discharge. She entered hospital on stretcher, May 10.

Physical examination. Patient is a female of moderate stature, poorly nourished, with typical hippocratic facies. The scalp is negative. The eyes react to light and accommodation. The teeth are in good condition. The tongue is very dry and badly coated. She has moderate enlargement of thyroid. The chest is negative except for a few coarse râles over the right base. Heart: apex at fifth interspace, nipple line, no demonstrable murmurs present. Pulse is very fast, quality poor, and equal in both wrists. Blood pressure 115–70. There is liver dullness from fifth rib to costal border. The abdomen is markedly distended, the bowel coils perceptible on respiration, and tympanitic throughout. Moderate tenderness is present over upper abdomen but extremely so over hypogastric and both inguinal regions. Vaginal examination shows second degree tear in perineal body, the parts are bathed in pus, and there are some small lacerations of the cervix. Bimanually, the uterine body is large and boggy, the posterior surface brawny and boardlike to touch, and there is no demonstrable fluctuation. The tubes and ovaries are not palpable but marked tenderness is present over both regions.

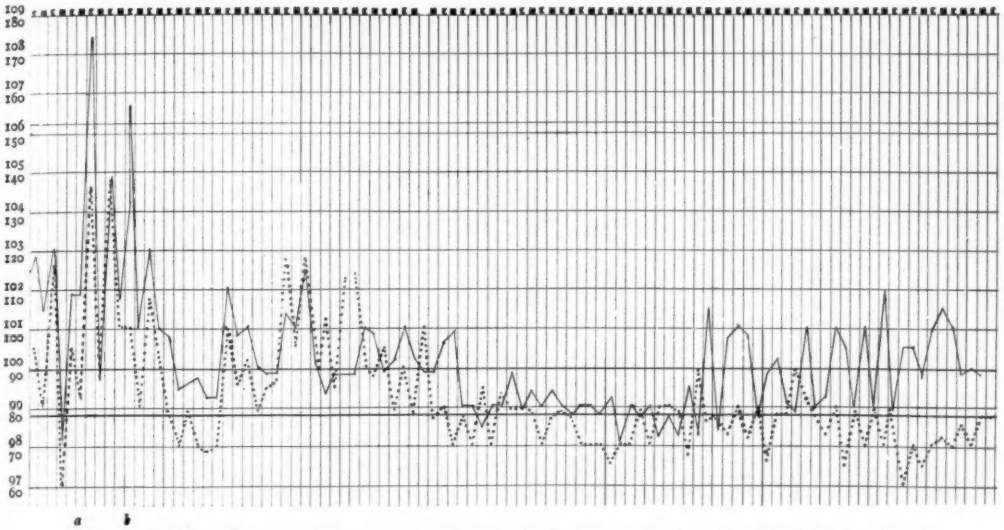

Chart 1. Pulse and temperature curves. *a*, Four hundred cubic centimeters citrated blood transfused; *b*, lymphaticostomy. Pulse——; temperature .....

Laboratory findings. White blood cells, 51,000; differential: polymorphonuclears, 96 per cent, small lymphocytes, 3 per cent; large mononuclears, 1 per cent; red blood cells, 3,336,000; hæmoglobin, 57 per cent; color index, 0.8 per cent. Urine from catheterized specimen shows specific gravity 1.020; acid; trace albumin; no sugar. Microscopic examination shows: few hyaline and granular casts, erythrocytes with some pus cells and bacteria. Stained smear from cervix showed some extracellular, gram negative cocci with some pneumococci.

Diagnosis: puerperal sepsis with general peritonitis. Course: May 11, 1923. Patient given 500 cubic centimeters of 10 per cent glucose intravenously. This was followed by chill lasting 30 minutes. The abdomen was badly distended, tympanitic throughout, and general condition about same as yesterday. Blood culture taken.

May 12, 1923, 8 a.m. General condition seems some better. She has no pain, tenderness on palpation is not so marked, there is no vomiting, and no dullness in flanks. Five hundred cubic centimeters 10 per cent glucose was given intravenously followed by a slight chill lasting 15 minutes. 5 p.m., intravenous glucose repeated with condition about same as in morning. White blood cells, 27,200; polymorphonuclears 96 per cent; small leucocytes, 4 per cent; blood type, 4. Needle inserted into lower right pleural cavity and into liver region with negative results.

May 13, 1923, 1 p.m. Patient given 400 cubic centimeter citrated blood—donor and recipient being same type. She had severe chill lasting 30 minutes but reacted well in spite of her poor condition.

May 14, 1923. Condition about same. White blood cells, 24,000; polymorphonuclears, 90 per cent; small lymphocytes, 10 per cent. Operation under 1 per cent novocaine. A 3-inch incision was made over the posterior border of the sternomastoid on the left side. Blunt dissection was carried down to the internal jugular which on separation from its sheath and traction medially presented the thoracic duct which was opened and drained after the method of Costain. Culture and smears were taken from thoracic fluid. Skin and fascia were closed with interrupted silkworm and hot hychlorite dressing applied. Dressing was changed in afternoon, draining profusely. Blood culture negative.

May 15, 1923. The abdomen is much softer, there is no rigidity, some decrease in size but there is a profuse drainage from neck. Four hundred fifty cubic centimeters 10 per cent glucose was given intravenously, followed by no chill.

May 16, 1923. Condition seems better, profuse drainage from neck, wick removed. White blood cells, 43,000; polymorphonuclears, 95 per cent; small lymphocytes, 3 per cent; large mononuclears, 2 per cent; red blood cells, 3,532,000; hæmoglobin, 80 per cent; color index, 1 per cent.

May 17, 1923. Drainage not so free, abdomen markedly decreased in size, no areas of tenderness or rigidity. Patient doesn't appear nearly so toxic, the tongue is moist, the extremities warm. She was given 500 cubic centimeters of glucose. The pulse is regular and of good volume. Smears from the thoracic fluid show gram positive diplococci (probably pneumococci). White blood cells, 33,400; polymorphonuclears, 90 per cent; small lymphocytes 8 per cent; large mononuclears, 1 per cent.

May 18, 1923. Drainage from neck very much reduced. The abdomen is nearly normal in size with no areas of tenderness or rigidity. Examination of culture of fluid from thoracic duct reveals pneumococci.

May 22, 1923. Patient is steadily improving, has no neck drainage, tympanities are nearly gone. She is hungry and is allowed semi-solids, is not nauseated, does not vomit.

June 4, 1923. Patient feels fine, the neck wound is gradually healing in by granulation. The abdomen is soft with no areas of tenderness.

July 4, 1923. Discharged. The neck wound is entirely healed, the abdomen shows no distention or areas of tenderness. Vaginal examination shows some discharge. Bimanual examination shows body of uterus contracted and freely movable with no pain on pressure over either the tubes or ovaries which are not palpable.

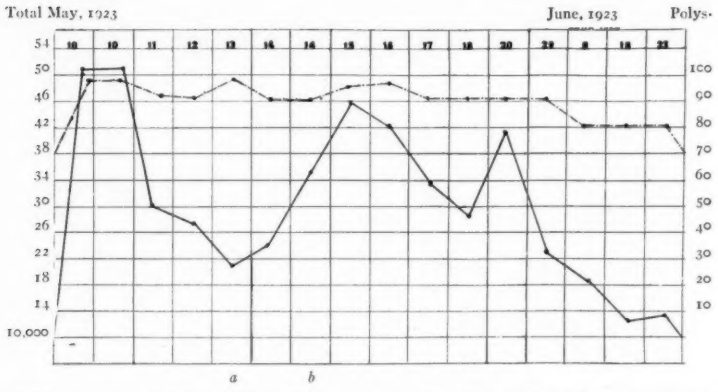

Chart 2. Total leucocyte and polymorphonuclear curve. *a*, Four hundred cubic centimeters citrated blood transfused. *b*, Lymphaticostomy. Leucocytes ——; polymorphonuclears —·—·—·—.

July 27, 1923. Patient feels well. She complains of no pain or distress of any kind. The abdomen is negative on examination. There is small amount vaginal discharge. Bimanual examination shows the uterine body small and freely movable. No pain or tenderness is present on pressure over the tubes and ovaries of either side. Blood examination: white blood cells, 7,600; polymorphonuclears, 71 per cent; small leucocytes, 21 per cent; large mononuclears, 3 per cent; eosinophiles, 5 per cent; red blood cells, 3,544,000; hæmoglobin, 63 per cent; color index, 0.9 per cent.

CONCLUSIONS

1. Recovery of the same type of organism from both cervical canal and thoracic fluid proves that a great deal of absorption takes place through the lymphatic system.

2. Lymphaticostomy is a relatively harmless procedure.

3. Drainage of the thoracic duct does relieve the toxæmia and in conjunction with other measures is a useful adjunct in the treatment of systemic type of puerperal sepsis.

I wish to express my sincere thanks to my associate, Dr. R. D. Thompson, to Dr. C. E. Myers, and to the Sisters of St. Mary's-Ringling Hospital, who made it possible for the presentation of the foregoing article.

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THE ISOLATION OF THE SUBMUCOSA AS AN AID IN INTESTINAL ANASTOMOSIS[1]

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THE purpose of this paper is to show the part played by the submucosa in the development and performance of enterorrhaphy; to explain, by directing attention to its physical properties, certain measures that are now being taken, such as walling off infected areas with bowel; and to suggest further utilization of this membrane.

The existence of the submucosa as an entity has been known many years, but an appreciation of it in intestinal suturing has not been sufficiently dwelt upon, the task being left for the individual surgeon to work out for himself. This lack of clearness was due, in part, to the older histologies, such as Stricker's (1), 1872, describing the bowel wall as consisting of two tubes, (1) mucous and (2) muscular. Its location and character were mentioned but no idea was given of its appearance as isolated; it lost its identity in the mucosa. The sliding of coats, or eversion of mucous membrane at operations occurs between the submucosa and muscularis. It was formerly customary to speak of the everted portion as "mucosa," and thus the submucosa was lost sight of.

HISTORICAL

To understand the development of this appreciation of the submucosa, it is necessary to review the estimates put upon it by surgeons of the past in intestinal anastomosis.

Lembert (2), in 1826, showed that healing must be by peritoneal approximation, and described the plastic lymph which seals the joint. His stitches penetrated "as far as" the mucous membrane. Operators of that day, thinking of the eversion of the mucous membrane, and of the "two tubes" often missed the submucosa entirely. Lembert said that his stitches encysted and were not sloughed into the lumen of the bowel. The researches of Schmidt, Thompson, and Travers soon afterward, however, showed that stitches as foreign bodies were cast off to the lumen of the bowel.

Fifty years later, or after abdominal operations were becoming more frequent, Czerny (3), in 1881, wrote, "in order to prevent the escape of intestinal contents, and to place the parts in an ideal condition for repair," he caught up the mucosa and (submucosa) in another row of stitches.

Lister (4), in 1881, first described the isolation of the submucosa, and brought the first notice of it to surgery by the following description: "Catgut, as you are doubtless all aware, is prepared from the small intestine of the sheep. The gut is treated in what seems an exceedingly rude manner for so delicate a structure. It is scraped with some blunt instruments, such as the back of the knife, over a board; and by this means, as the people express it, the dirt is scraped out. That which these people call the dirt is the exquisite and complicated structure of the intestinal mucous membrane. But while the mucous membrane is scraped out from within, there is also scraped off from without the circular coat of muscular fibers. The result comes to be that the intestine is converted into a comparatively unsubstantial material consisting of two parts or bands, one more slender than the other. When the mesentery is stripped off by the butcher the peritoneal covering of gut shrinks into a narrow strip, and this, with some of the longitudinal fibers, constitutes the more slender of the two parts to which the intestine is reduced by this process of scraping. The

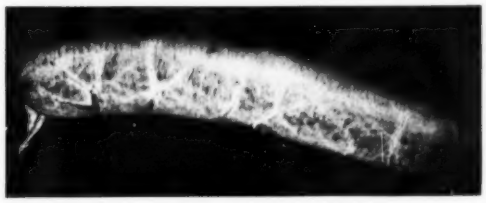

Fig. 1. Photograph of sausage casing. Observe its deviations under the blood vessels.

[1] Read before the Chicago Surgical Society, May 4, 1923. (For discussion, see p. 278.)

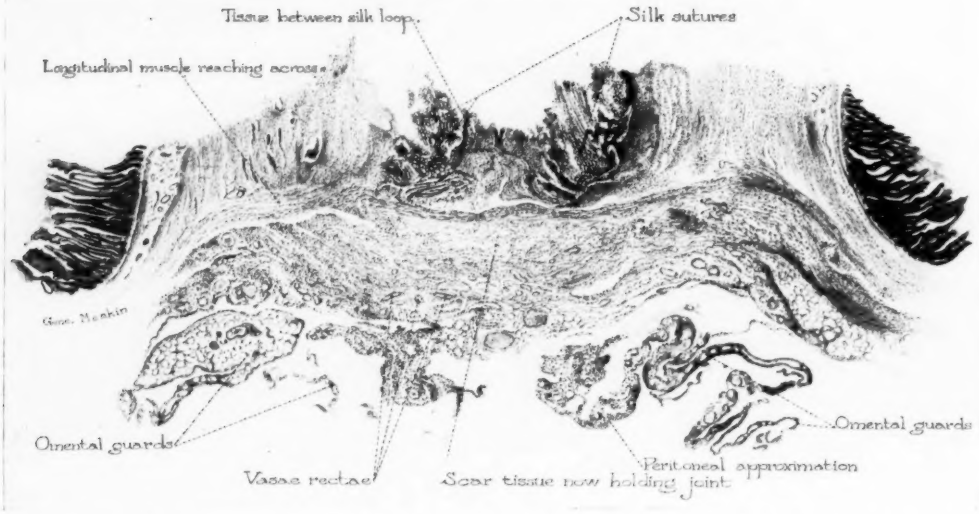

Fig. 2. Five day joint. Note loops of silk sloughing out; the reaching across of longitudinal muscle; scar tissue holding the joint at this time; remains of two vasæ rectæ which were placed side by side at the operation; and the omental guards.

other part is the essential material from which catgut is prepared, and this is neither more nor less than the submucous cellular coat of the intestine. When I first visited a catgut manufactory I was astonished to find that, after this scraping process, the intestine could be blown up still as a continuous tube, as you see can be done with this specimen, which has been treated in the manner I have described. This exquisitely delicate structure is a beautiful anatomical preparation of the submucous cellular tissue, though made in so rude a fashion. This coat of the intestine, which, in the sheep, has this extraordinary toughness, is the material out of which catgut is prepared."

Bacteriology at this time cleared up the question of external sepsis so that causes of failure could be understood.

Mall (5, 6) demonstrated in 1887 the reticulated tissue in the stratum fibrosum.

W. S. Halsted (7), in 1887, and working later with Mall, isolated the submucosa and described it as "air-tight and water-tight." Employing needles with dulled points he proved that sutures with a grasp of the serosa and muscularis only were "not to be trusted." (Exp. A, Halsted, 7.) He insisted that "each stitch should include a bit of submucosa" and stated, "I am not aware that the importance of this coat has been hitherto emphasized." Again, in 1891, he wrote, "Success depends upon an appreciation of the importance of the submucous coat of the intestine," and that it was "remarkable that surgeons could have overlooked the existence of the submucosa"; and again, "About 3 years ago I endeavored to emphasize the importance of the submucous coat in operations, but succeeded only in attracting attention to the quilt or square stitch." He feared then the entrance of the stitches into the lumen, and thought the stitches encysted, or sloughed to the serosa.

M. E. Connell (9), in 1892, "in order to reduce the number of stitches and knots" devised the continuous mattress suture.

Murphy (10), in 1892, with the button, demonstrated how quickly the intestinal coats healed (alluded to by Moynihan later). According to Barbat (11), the button pushed all the tissues out of its grasp except the submucosa and peritoneum.

Senn (12, 13), in 1893, wrote: "Halsted's advice to include in the stitches the firm fibers of this submucosa, is important and should

Fig. 3. Nine day joint. *a*, silk loop; *b*, ulcer closing; *c*, vasæ rectæ uniting.

Fig. 4. Sixty-one day joint—delayed union. *a*, limits of epithelium closing over; *b*, pyramid of exudate; *c*, silk cavity; *d*, dense scar tissue.

never be ignored"; and speaking of the Czerny-Lembert method said, "the inner stitches ulcerate into the bowel; the outer ones become encysted."

Edmunds and Ballance (14) of the Brown Institution, London, in 1896, quoted Lister's description of the isolation process, and showed that the sliding of the mucous membrane over the muscles took place between the submucosa and muscularis. They used both Lembert and Halsted stitches and proved that both sloughed to the lumen of the bowel. They thought it remarkable that in Maunsell's operation the circular suture of through-and-through unabsorbable material should leave fewer signs, in a given time, than the Lembert stitches in the longitudinal slit.

Spalteholz (16), in 1897, described the connective-tissue framework of the mucous membrane of the small intestine of the dog as consisting of a dense network of reticulated, collagenic and elastic tissue.

H. A. Kelly (17), in 1898, wrote that "the most valuable contribution which has yet been made to intestinal surgery is the demonstration by Dr. W. S. Halsted of the fact that the essential feature in any suturing or anastomotic operation is the employment of the submucous intestinal coat," and referred to its other applications by comparing the relative thicknesses of the submucosa in the large and small intestines as being of the ratio of 4 to 1 respectively, and by utilizing the sigmoid in walling off pelvic infections by placing it around the brim of the pelvis.

OBSERVATIONS AND EXPERIMENTS

In an attempt to secure for the students in the College of Medicine of the University of Illinois the appreciation of the submucosa in experiments in intestinal work, as well as to investigate its physical properties, its study and isolation were begun.

In the first place, everything made from the submucosa was secured from the packing house, as catgut, tennis-racquet and violin strings, and Cargile membrane (derived from the trachea and now called Allison-Brooks

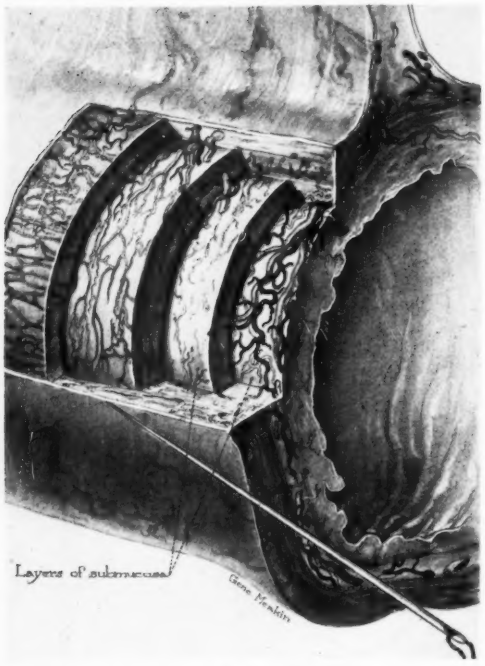

Fig. 5. The coats in echelon with needle applied for the Lembert stitch and controlling hæmorrhage.

membrane). The manufacture of most of the articles was observed. This gave an entirely different view of the relative strength of the various layers of the bowel wall and it was readily seen why some stitches pulled out, and why care was necessary in taking the Lembert stitch.

The submucosa was studied in six ways:

1. Raw bowels. Sections of beef bowels were used in practicing the fundamental stitches and in making unions. Fortunately, at one of these periods sausage casings were sent, and the students were obliged to familiarize themselves with this vital layer—the submucosa.

2. Dogs. Experiments on dogs were made as follows: The submucosa was exposed by removing the overlying coats at the pylorus and at various places in the small bowel, the muscles being the easier detached as the ileum was approached.

3. Excursions. The class was taken to the Chicago Stockyards, for it is only from watching the packing-house prepare sausage casings and catgut that this tissue assumes its identity. There, after macerating the bowel in water for a time, it is pulled through the "slimer," which removes mucosa, muscles and peritoneum, leaving the submucosa, a slender translucent shred, or a casing for sausage (Fig. 1). That it is a permanent layer may be seen as it deviates under a blood vessel as a subway runs under a river, leaving the course of the vessel outlined as it branches toward the antemesenteric border.

It will prove highly instructive, as well as a time-saving process, for the surgeon to visit an abattoir and see the bowel macerated and stripped. Afterward, he will not allow the submucosa to slip from his line of suturing, as sometimes happens in practice on animals. The unpleasantness of this work in their attainment of asepsis has kept surgeons from this study heretofore. They should see (1) the maceration and stripping of the muscularis and mucosa; (2) the distention of casings with water; and (3) the spinning of the submucosal strands into catgut.

4. At operations. Dr. Alfred A. Strauss in demonstrating operations for pyloric stenosis, pyloric closure, and repair of ulcers, made the following manipulations: (1) He stripped the muscularis from the submucosa in very much the same manner as one would peel a banana; (2) he threw a band of rectus fascia between the submucosa and muscularis at any point desired for a closure of the lumen, at the pylorus or elsewhere; (3) he even patched with rectus fascia the opening made by a gastric or duodenal ulcer. The submucosa formed the buttress of his suture spans. In all this, the thing that impressed me was the way the submucosa stood out as the "skeleton" of the bowel structure. The conclusion followed that this tissue was not sufficiently emphasized in abdominal surgery.

5. Microscopic comparisons. Histological sections of sheep's gut were compared with its product, catgut, teased out and stained; that of normal hog gut with its product, sausage casing, stained, noticing that in the hog gut slide the submucosa almost loses its identity as a layer under ordinary staining methods.

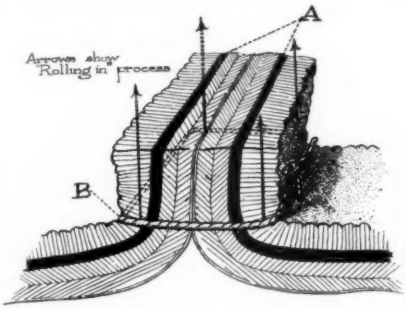

Fig. 6. Typical stitch with submucosa emphasized. Diagrammatic. Showing the "rolling-in" process.

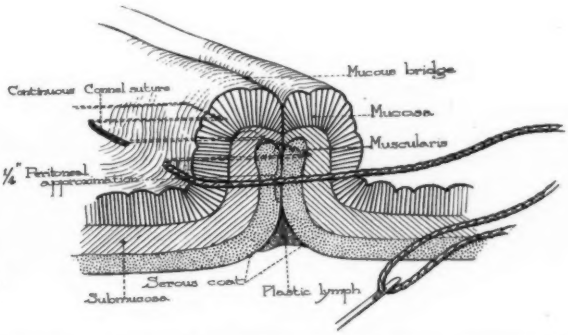

Fig. 7. The continuous Connell suture, showing grasp of submucosa and approximation of peritoneum.

6. Regeneration of layers. To verify the permanency of its tissue by the slowness of its regeneration, junctions of the jejunum in the dog were made. Sections were made parallel to the lumen of the bowel, the fate of the silk sutures noted over periods of 5, 7, 9, 31, 59, and 61 days and the regeneration of layers traced. The silk fibers swelled up and yielded easily to the sectioning knife in any plane. The silk particles of some of the later joints seemed hard and brittle, taking the stain badly, and breaking up on the edge of the knife. The silk always acted as a foreign body as shown by its migration toward the mucous membrane to be cast off (Fig. 2).

The silk loop was almost sloughed out in 5 days, the scar tissue holding the joint, but fragments of silk were found as late as 31 days. In the 59 day joint the site could only be identified microscopically by a few inflammatory cells, and by one layer of the muscularis mucosæ being somewhat thickened. Of course, the submucosa was completely regenerated (Figs. 3 and 4).

PROPERTIES OF SUBMUCOSA

From these and other sources the submucosa may be described as follows: isolated, it appears as white translucent strands.

Histology. Carey (18) tells us that the submucosa is arranged in two spirals, the inner one making a complete turn in 0.5 to 1 millimeter and the outer one in from 4 to 10 millimeters. The submucosa takes only the diffuse stain, which accounts for the fact that the student loses sight of its importance surgically. He should recall from histology white fibrous connective tissue; and then examine the drawings of reticulated tissue, for, doubtless, the makeup of the transcluent strands is composed of (1) the reticulated, collagenic and elastic tissue of the connective tissue framework of the mucous membrane, (2) the muscularis mucosæ, and (3) the submucous fibrous and elastic tissues.

Strength. Goldbeaters' skin, a derivative of the peritoneum which is a much thinner structure than the submucosa, in books of 1,000 leaves making 1 inch in thickness, withstands the beating of a 6 to 16 pound hammer 10 hours a week for a period of 2 years—a test of its permanency—yet it will tear in one direction as easily as paper.

Thickness. The tracheal submucosa (weasand) varies from one-sixteenth to one-hundredths of an inch—the thinnest Cargile membrane. It would probably grade for the different organs as follows: trachea, large intestine, small intestine, ureter, fallopian tube, pelvis of kidney, gall bladder, and gall ducts.

Resistance to the needle. As the needle engages it in taking the Lembert stitch, a point ahead of the needle where the blood is pressed out of the muscularis and serosa forms the "white ischæmic spot." It should give the resistance of rubber dam. A *pause* should be made to get these tests in taking the Lembert stitch, and the needle not be rapidly thrust through the bowel, as a Hagedorn is entered through muscle or fascia. Practice only will

educate the operator as to when and how his needle penetrates the submucosa (Fig. 5).

Permeability. a. To gases. Goldbeaters' skin is the only known membrane that is impermeable to hydrogen gas for any length of time; hence its use in airships. That the bowel wall has such powers is evidenced by its being capable of retaining gas under the enormous distention of tympanites in certain cases of obstruction.

b. To liquids. Normal salt solution easily passes by osmosis through the sausage casing.

c. To bacteria. The membrane is not permeable to most bacteria. Bacteriologists tell us that the lymph glands on the mucosal side are practically always infected, and the fact that peritonitis is not more frequent proves what a barrier the submucosa is. Rammstedt, in his operation for congenital pyloric stenosis, took a forward step when he relied upon the submucosa to keep back the bacteria from the peritoneal cavity with the assistance only of the oversewed omentum.

d. To toxins. The membrane is not permeable to toxins of large molecular size. It will retain the ptomaines in an obstructed loop, or in ptomaine poisoning, and even fatal cases are accompanied by a very tardy development of peritonitis. That it acts as an obstruction to drainage is shown by the more frequent practice of opening infected loops in obstruction. Sala (19) had 7 such cases. And to avoid such a condition, surgeons, fearing the effect of such retention and pressure on the junctions, are opening the bowel to the exterior below (Hohlen, 20). Where paresis of the bowel can be demonstrated, it seems that the making of an artificial fistula is indicated. Natural fistulæ are certainly rare compared with the number of cases of peritonitis.

The function of the submucosa is, therefore, three-fold: it is (1) connective (2) supportive, the "skeleton," and (3) retentive. These functions are recognized by the resistance to pathological processes, the rarity of fistulæ, and the slowness of regeneration. It is retentive to gases, bacteria, and toxins, as in obstruction from tuberculous peritonitis with enormous distention, or to ptomaines developed from poisoning or from an obstructed loop.

Applying this impermeability to the walling-off process, Privat (21), in 1846, not being satisfied with Lembert's method closed four perforations of the intestine by suture. When this failed, he secured closure by placing an adjacent healthy loop against each perforation, and concluded that that was a better method than suturing.

The absence of the submucosa in the uterine mucous membrane is a factor in the explanation of the immunity of the pelvic peritoneum.

RÉSUMÉ

Returning to the part played by the submucosa in intestinal suturing, Figure 6 represents diagrammatically a unit or typical stitch of the Connell mattress suture. Lifting up the everted mucosa and submucosa, the latter presents as a pale, shining membrane like the raw side of leather (landmark). The needle penetrates the united coats, catches the retracted muscles, secures peritoneal approximation with the opposite side, picks up its submucosa, passes over a space of mucous membrane, and returns to near the point of entrance, and is tied. The connective tissues form two splints: *a*, layers of submucosa, and a bandage, *b*, (suture material) compressing the soft yielding tissues, the glandular mucosa, the vascular muscles, and peritoneum. (Fig. 7).

The method of union is very simple as shown by Moynihan (22) as follows: "But I still think that the great virtue of the button (Murphy) was not in its own use, but in the convincing demonstration it gave to the essential simplicity of the process of visceral union. By using the button we learned how safely and how rapidly the peritoneal junction took place; there was no need, it was now perfectly evident, for the hundreds of stitches that all surgeons were using. Firm, even approximation for a very few days would lead, the button showed beyond a doubt, to permanent and secure fusion of the apposed viscera."

The intestine is very vascular as a tissue, with free longitudinal anastomosis in the submucosal vessels. Within an hour the peritoneal junction is covered with plastic lymph, which the broken circulation throws out as an expression of the injury suffered. The healing process has begun. This plastic lymph should not be disturbed by manipulations, nor allowed

to be liquefied by bacteria from the mucosa. It will bury the stitches, if they are pulled taut, from the peritoneal surface, and no "capillarity" can take place, the stitch (foreign body) being immediately on its way to the mucosa to slough out. This "rolling-in" process of sloughing of suture material is complete, for the most part in 5 days (Figs. 2 and 6) which might be called the time of repair.

All recent bowel operations show that the importance of this coat is appreciated. This is especially true regarding the Rammstedt (23) operation for congenital pyloric stenosis, the operations upon small ducts—anastomosis of the ureter, and anastomosis and transplantation of gall ducts—which were done in the experimental laboratory a long time before surgery adopted them, and recently, the aseptic operations for the resection of bowel by Collins (24), and Horine (25). Suturing over clamps is inconceivable without a knowledge of the integrity of the submucosa. As Halsted applied the method upon the heavy submucosa of the rectum and sigmoid, areas in which it is impossible to use the Connell suture because of their inaccessibility, there seems to be no reason why it should not be applied to the bowel high up.

CONCLUSIONS

1. In Lembert's time the principle was established that "raw tissues of like nature will unite if held approximated long enough" and union was secured by approximating the peritoneal coats.

2. Halsted showed that all intestinal suturing must be with the submucosa as the basis (the skeleton).

3. Students should see this structure isolated; they take better stitches afterward.

4. The submucosa must be taken into consideration in regard to drainage of bacteria and toxins from obstructed loops of bowel; and in regard to making and closing fistulæ. It may be utilized to prevent extension of peritonitis by walling off areas.

5. All through-and-through and most Lembert stitches slough to the lumen of the bowel; a few encyst at the serosa, being cut off by the action of the muscular coats.

6. The tendency to throw off a foreign body by sloughing out is stronger than the tendency to absorb it.

7. Visceral union is a matter of sloughing of suture material, and of absorbing superfluous tissue, and is accomplished in a few days.

8. From the study of this tissue there has been evolved the aseptic resection of bowel by Halsted and others.

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ABDOMINOSCOPY

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ABDOMINOSCOPY is a term which describes our new method of examining the organs of the abdominal cavity, in reality an endoscopy of the abdominal cavity. As far as we know abdominoscopy has never before been performed on the living patient or on the cadaver. Also I have been unable to find any papers dealing with the experimental or theoretical phases of the subject. Therefore I believe that it is so far an unknown but practical method of examining the peritoneal cavity and that I am justified in calling it a new means of diagnosing abdominal diseases.

Not only the common practitioner but the specialized diagnostician as well would in many cases feel more assured if he could but look into the peritoneal cavity. Many methods, some of which are complicated, of examining the abdominal organs have not proved successful and consequently after their use we are no wiser than before. It would be an ideal method of examination if we could look into the peritoneal cavity through a puncture and, without doing a laparotomy, see the diseased organs clearly and sharply.

The first cases in which we attempted the method were ascites cases in which paracentesis was indicated. An endoscope was put into the peritoneal cavity, but we were able to obtain only practically negative results. We next decided to inflate the abdominal cavity of the cadaver with gas, and to study the conditions with the endoscope. Theoretically this seemed only partially feasible. Sometimes we were able to see clearly but on the whole the examination of the cadaver was not satisfactory because of postmortem changes.

We tried out experiments on bodies immediately after death and found that our assumption was correct; abdominoscopy was not only possible, but yielded results far beyond our expectation. The whole problem was solved at once. A technique for practical use on the living body was perfected and our endeavors were far more successful than we had obtained on the fresh cadaver, as physical conditions for carrying out the abdominoscopy were more favorable.

INSTRUMENTS

a. Abdominoscope. The instrument devised resembles the cystoscope. As the curved end of the cystoscope was found to be very useful, it was made movable. The degree of curvature was regulated by a special mechanism at the opposite end of the instrument. To introduce the instrument with ease, we were careful to regulate the curvature at the end so that it was in the axis of the endoscope.

While examining the cavity, by extreme flexion the top of the curve could just be seen in the periphery of the endoscopic field, so that we were able to use the top as a guide. The curve at the end of the instrument is of prime importance in keeping from the endoscopic field the different structures, such as the liver edge, etc. Thus, to a certain extent we can use the top as a *tractor*. Another important use of the end is as a palpator to determine the consistency and movability of the organs and the presence of gall stones. The magnification used in the telescope should correspond to that used in a cystoscope, since a great magnification only makes the survey difficult and offers no advantage. The telescope should be exchangeable so that it can be introduced with the obturator and making it possible to remove it to clean it during an examination. The endoscope has a canal for the introduction of gas. This canal is similar to that used in a cystoscope for irrigation purposes. By means of this canal the gas is made to escape under control of the eye into any pockets. The air cock of the endoscope is connected to a bulb similar to that used on a blood-pressure apparatus. A 12-inch rubber tube which connects the air cock to the bulb is divided by a glass tube which contains sterile cotton for cleaning the gas. For examining the pelvis we recommend an endoscope with a direct outlook, but this is not necessary.

Examination can be made also with a cystoscope but the cystoscope does not give as satisfactory results. When the cystoscope is used, the right hand cock for irrigation is connected with the connecting rubber tube, and the left hand cock must be closed. The cystoscope must close airtight.

b. Trocar. The trocar must correspond in thickness with the abdominoscope. For the cannula for the trocar we use only *half a tube.* A larger trocar would make the introduction easier but would endanger the keeping of the belly air-tight. In using a cystoscope it is best to take a tube of thin, flexible metal, which is open in the whole length on one side. It will then be easy to bring the cystoscope through the slide-way into the abdominal cavity. When the trocar and cannula are removed together, it is usually possible to introduce the cystoscope through the puncture canal, but it is safer to use a tube as described, especially if the patient is fat.

c. Gas for inflation. At first we used oxygen for better absorption. Now we are using the atmospheric air, and have had no ill effects. When the examination is finished, we *slowly* let out most of the gas, so that there will remain only a small amount of the air in the belly. The use of the air, cleansed by the filter mentioned has essentially simplified the whole procedure and has made possible the direct inflation with the bulb. It is essential also to sterilize the connecting rubber tube. At first we measured the quantity of air used, but we have found that this is unnecessary for the abdomen is not very sensitive to inflation and easily withstands the quantity of air necessary for abdominoscopy.

TECHNIQUE

A purgative is given the day before examination and one-quarter grain of morphine 20 minutes before puncture. The field of puncture is cleansed with benzine and then with alcohol and iodine. Local anæsthesia with novocaine is used, and it is a good plan to anæsthetize also the adjacent peritoneum to prevent sensations during examination.

A stab incision is made through the skin and a puncture done with the trocar inserted. Puncture should always be made with the abdominal muscles contracted. Usually there is a reflex contraction; if none is present, we let the patient sit upright. The puncture must be carried out steadily and cautiously. To prevent deep entrance into the abdominal cavity, the trocar should be carefully steadied with the left hand while the abdominal wall is being penetrated. Carelessness in penetrating the abdominal wall must be avoided. The trocar is removed and the abdominoscope is introduced along the tube which remains in the puncture canal as has been described. Then we remove the tube from the puncture canal. This is necessary for an air-tight closure.

The abdominal cavity must be inflated slowly. This can easily be done with the bulb. At first we were astonished to find how little resistance the abdominal wall offered to the inflation. More surprising was the little effect the inflation had on the patients. When the examination is finished, we must allow the air to escape slowly so as to prevent disagreeable sensations. We remove the endoscope and with the hand placed flat on the belly we press out what air remains. The wound is closed with one suture, swabbed with iodine, and a small dressing applied.

In most of the cases we made the whole examination using local anæsthesia. Some patients are alarmed when the trocar is introduced, but they complain of no pain even when a total abdominal examination is being made. We often keep them so interested in our description of their organs that they are quite amused.

ABDOMINOSCOPY

Of first importance in performing abdominoscopy is the original *position* and the correct changes of the position of the patient. As the air is of light specific gravity, it stays uppermost in the abdominal cavity. Therefore, through changes of position of the patient we are able to put the air in any place and thus displace the intestines at will. With the patient in a horizontal position, by moderate inflation, we have a full view of all organs in their normal relation under the abdominal wall. For an examination of the upper part of the abdomen, we elevate the

thorax above the horizontal. For an examination of the pelvis, we change the position of the patient so as to elevate the pelvis above horizontal. If we want to examine the organs in the left side of the abdomen, we put the left side uppermost.

Therefore it is evident of how great importance is the position of the patient for the success of abdominoscopy. We need a safe and easily *changeable table* for the examination.[1]

The abdominal cavity is inflated only moderately at first. If more air is needed during the examination or if the belly is not airtight, more air is easily gotten by compression of the bulb with the right hand. In this way at any time during the examination, we are able to regulate the degree of inflation. Also it is possible under full view to bring air into pockets, between bowels, etc.

The endoscope should be moved only under control of the eye.

Internal palpation. This palpation with the endoscope is very useful for determining the consistency and movability of organs and for sounding for gall stones. Internal palpation was found to be the most important item in the whole examination, and it was the main consideration in the construction of the abdominoscope.

Systematic examination. The whole examination should be done with a fixed plan in mind; otherwise the wonderful natural pictures would tend to lead astray and thus prevent seeing important points.

In passing we might say that it is not advisable to look immediately for the suspected organ. At first we see a large endoscopic field which is increased in magnitude by moving the endoscope in different directions. After we have become familiar with the anatomical pictures of the region we then examine the diseased organs.

After a few examinations with the endoscope, the pictures become so clear and natural that they will be very easily understood.

Site of puncture. We try to make one puncture do for the examination of the whole peritoneal cavity. From a point slightly below and to the side of the umbilicus, it is possible to see a good deal of the organs in the upper part of the abdomen, also we can rotate the endoscope so as to observe the region of the symphysis. Usually we know in advance the region where the pathology is located and choose a puncture point near by, since this allows a better examination. Usually we avoid the mid line, and prefer the rectus muscle for the puncture, for the muscle has the advantage of a better closing of the puncture canal.

[1]With the assistance of C. Lewis, B. Huey.

FIELD AND RESULTS OF THE METHOD

When the puncture is made near the umbilicus, with patient in the horizontal position, we can look over the greater part of the abdominal cavity. The intestines and the omentum are usually found to be in one plane. The intestines show a hilly arrangement and peristalsis can be observed. We are able to see, on either side to the lateral abdominal wall, below to the symphysis, and above as high as the diaphragm. The nearness of approach to the different organs depends only on the length of the endoscope.

Puncture above the umbilicus gives a surprising survey over the upper part of the abdomen when the thorax is elevated. The liver is so far away from the abdominal wall that we can examine the surface to a great extent. On the observer's left we can see as high as the vault of the diaphragm. The top of the gall bladder, if not visible, can be demonstrated by turning the patient slightly to the right side so that the liver edge can be easily lifted by the abdominoscope. Usually a good examination of the gall bladder can be made. With the abdominoscope we can sound for gall stones if the gall bladder is not too tense.

With the same procedure a good deal of the upper and anterior part of the duodenum and of the pylorus can be demonstrated. Also there is a beautiful view of the uncovered part of the stomach. Palpating between the stomach and liver with the endoscope, other parts are brought into view. By inflating the stomach per os, the greater part of the anterior surface of the stomach can be examined. The normal spleen can usually be seen in the region of the hilus, sometimes

the convex surface is far removed from the diaphragmatic wall. We were often surprised to find the spleen far up on the lateral side.

Puncture in the lower part of the abdomen with the pelvis greatly elevated shows a beautiful endoscopic picture, the first view of which was like the fulfillment of a dream; the whole pelvis lay free and unobstructed before the eye. The uterus, tubes, ovaries, and sigmoid could have been shown very little better by laparotomy. Elevating the right side we were able to inspect the cæcum, especially when puncture was made at McBurney's point. Not so rarely it is possible to see the appendix.

When we made it possible to survey under direct vision the abdominal cavity *in situ*, the great importance of this method of examination in abdominal diseases became evident. We were not only able to see the organs far more clearly than we had expected, but through changing the position of the patient the endoscope revealed new and important regions. When examining the abdominal cavity for the first time with the endoscope a great surprise is in store for us for we see the different organs in their natural, living colors.

The value of abdominoscopy lies in its ease of application and the marvelous results obtained through the direct, eye-controlled method of examination. In this paper we will not enter into the differential diagnostic possibilities; through the abdominoscope we believe it will be possible to diagnose the questionable case correctly and without delay. We would call attention especially to the importance of its use in making an earlier and surer diagnosis, thus making it possible to decide earlier as to the advisability of operation in cases showing grave pathology, such as metastasis of the liver, etc. The indication for its use in gynecological cases will need no further comment when the profession realizes the extent to which this ideal method of examination of the pelvic organs can be carried out.

The method used is not difficult. The technique may seem complicated but it is just as easy to carry out as is cystoscopy. The abdominoscope is easily introduced. The aseptic preparation is scarcely more exacting than for puncture in ascites cases. The whole method is simplified by using cleansed atmospheric air, which is forced directly by means of the bulb through the abdominoscope. In some cases examination can be carried out with a cystoscope, but this is not so satisfactory. The examination can easily be made under local anæsthesia without pain to the patient. Puncture with the trocar in place, if done carefully and the abdominal wall is contracted, is without danger. Atmospheric air did not prove to be disadvantageous; we did not have shock or any other complication in our application of the method. As a rule temperature and pulse remained the same. The patients were up and about the next day after examination. Pain at the site of puncture is unimportant. The general condition remains the same as before examination.

SUMMARY

The introduction of gas into the abdominal cavity has not only made endoscopy of the peritoneal cavity possible but has made vision very clear.

The abdominoscope is introduced through a puncture made with a trocar, thus making it possible to view the peritoneal cavity; in other words, abdominoscopy is similar to cystoscopy. The method is not difficult, is not dangerous and does not require a special amount of skill. The examination can easily be done under local anæsthesia.

According to our practical experience abdominoscopy is a direct, ocular method of great practical use in the large field of abdominal diseases.

A questionable diagnosis can often be excluded or confirmed and a decision reached as to form, kind, and extension of pathology. We would emphasize especially the importance of this method which makes it possible to make an early and sure diagnosis both in general surgical conditions and in gynecological conditions.

The very practical results of this relatively simple method of abdominoscopy will command for it a place similar to that now held by cystoscopy.

EDITORIALS

SURGERY, GYNECOLOGY AND OBSTETRICS

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FEBRUARY, 1924

THE TREATMENT OF ACUTE INTESTINAL OBSTRUCTION

DISCUSSING this subject some 15 years ago with a well-known surgeon, he expressed the opinion that the treatment was by that time stereotyped, and that there was nothing further to be said on the subject other than to persuade physicians and general practitioners to refer patients suffering from acute intestinal obstruction to the surgeon at an early stage of the illness. This statement would have been true and would still remain true provided that we saw these cases within 24 hours of the onset of the trouble.

For over 20 years I have been in the habit of describing cases of intestinal obstruction as I have met them in three different stages, the treatment of each of which must differ. The first stage is that in which the patient is seen early (within 24 hours). His general condition is good and there is but little intestinal distention. The second stage is that in which the patient is not seen until later—2 days, 3 days, 4 days. His general condition is good, but there is considerable intestinal distention and severe vomiting which may or may not be stercoraceous. The third stage is that in which the general condition of the patient is bad. His pulse is feeble and perhaps intermittent, and vomiting is stercoraceous. The abdomen is greatly distended and the patient presents the appearance of one profoundly poisoned by the absorption of toxins, whether the result of bacterial activity, or a proteose intoxication, or, a combination of both conditions.

The treatment of the first stage consists in washing out the stomach with bicarbonate of sodium solution, after which a general anæsthetic is administered and the abdomen is freely opened. The cause of the obstruction is searched for and removed, after which the abdomen is closed. The stomach is again washed out and the patient returned to bed. Lavage of the stomach after operation is more important, in many cases, than it is before the operation.

In the second stage a similar procedure is adopted until the obstruction is discovered and removed. A separate incision should then be made through the left rectus muscle above the umbilicus, and a loop of the jejunum as close to its origin as possible is brought out through the wound. Into it is fastened a tube of 7 or 8 millimeter diameter after the method of Senn's gastrostomy except that only one or two purse-string sutures are used so that too much subsequent narrowing of the intestinal lumen may be avoided. The intestine is then returned within the abdomen and fixed with two catgut sutures, one on each side of the tube, to the parietal peritoneum and posterior sheath of the rectus. A lunated piece is cut out of each side of the end

of the tube introduced into the intestinal opening so that if it should accidentally impinge upon the opposite wall of the bowel the intestinal contents could still escape freely.

By this procedure the distended intestines will be allowed to empty themselves of their poisonous contents. The central wound is then closed. The stomach is thoroughly irrigated as before, and the patient put back into bed. An experienced nurse or a senior student is directed to continue irrigating the intestines with bicarbonate of sodium solution by siphonage through the tube. In this way without taxing the patient's strength and without producing any shock, the intestines are assisted to empty themselves.

This process can be continued for several hours, at the end of which time the entire intestinal area between the stoma into which the tube has been introduced and the point at which the obstruction existed will have been emptied of its contents, and these contents replaced in large measure by a fluid containing sodium bicarbonate and glucose, the absorption of which will counteract the tendency to acidosis and help to build up the reserve carbohydrate as well as replace the fluids of which the tissues have been deprived.

Impressed as I have been with the soundness and importance of the views enunciated by Mr. Victor Bonney of the Middlesex Hospital, London, in the *British Medical Journal*, I have discarded all other methods of emptying the distended intestines, and have followed the procedure outlined above.

The tube can be removed in 24 or 48 hours under gas or local anæsthesia, and the opening into the intestine closed by a single mattress suture. Dr. C. H. Mayo's suggestion of bringing the loop of intestine out through a hole in the great omentum may obviate the necessity of a suture for closure of the intestine after removal of the tube. Lactose may also be administered in large quantities, either by mouth or through the intestinal tube, as it has been shown that lactose can eliminate proteolytic bacteria from the intestinal flora.

In the third stage the patient cannot stand the administration of any general anæsthetic, and he is not even removed from the bed in which he lies. The stomach is washed out as before and a one-half per cent solution of novocaine should be used to infiltrate the tissues in the middle line above the umbilicus through which an incision is made into the abdomen sufficiently large to introduce the finger and withdraw a loop of the jejunum as near to its origin as possible. A tube is then introduced into this fluid-containing segment of gut and siphonage continued as before described. Should the patient survive, it may be possible to open the abdomen, seek out the cause of obstruction, and remove it at the end of a week. Meanwhile, nourishment can be given through the tube directly the poisonous intestinal contents have been evacuated, or it may be given by mouth and the tube need not be removed until the patient has recovered from the effects of the second operation.

I am convinced that in all cases of intestinal obstruction in which stercoraceous vomiting has occurred, drainage of the jejunum as close as possible to its origin should be instituted. This drainage can be assisted by repeatedly filling up with bicarbonate of soda solution and siphoning off the intestinal contents after the patient has been returned to bed.

As has been pointed out by Dr. J. E. Summers, of Omaha, Nebraska, Bonney's method of performing a jejunostomy is the one flaw in his otherwise sound paper. The treatment of acute obstruction engrafted upon chronic is somewhat different. The site of the chronic obstruction will almost invari-

ably be found to be somewhere in the large intestine, and in all such cases the bowel above the obstruction should be drained at once. A cæcostomy after the method of Sir Harold Stiles gives the best results so far as tiding the patient over his immediate dangers is concerned. This carries out Bonney's idea of draining the fluid-containing segment.

Until physicians and general practitioners can be educated to recognize that there is no treatment for intestinal obstruction other than early surgical interference, the mortality attending the treatment of these cases must remain very much where it is today—a mortality that is a disgrace to the profession. I believe that the mortality attending the treatment of such a condition as acute intestinal obstruction should not be more than 1 or 2 per cent, but such a consummation can only be attained by operating upon these patients within 24 hours or 36 hours of the onset of the trouble.

It may be noted that I have not attempted to deal with one very common cause of intestinal obstruction as it is a different subject and requires different treatment, namely, acute intussusception.

SIR WILLIAM TAYLOR.

MEDICAL AND SURGICAL CO-OPERATION IN CASES OF DIABETES AND EXOPHTHALMIC GOITER

IN making diagnoses, the benefit to the patient from the close co-operation of internist and surgeon, is too well recognized and practiced to need emphasis. The importance of similar close co-operation in the immediate pre-operative preparation and postoperative treatment of patients with metabolic diseases is not so generally practiced on account of the customary separation of hospital staffs into medical and surgical divisions, and these in turn into sub-groups, each with its own personnel, wards, and laboratory facilities. The result is often an unfortunate break in the continuity of medical co-operation in the first twenty-four to forty-eight hours after operation, and often even in the last twenty-four hours before operation. In certain types of disease, like diabetes and exophthalmic goiter, this transfer of a patient from the medical to the surgical ward at the moment his life is to be endangered, and the corresponding change in the responsibility for his care to a group untrained in the refinements of the medical management of these diseases, may be a very dangerous procedure. To carry out surgical procedures on such patients successfully requires careful judgment as to the time, type, or extent of operation, medication, and diet. The best results are possible only when there is no interruption in the expert medical supervision and treatment before the patient goes on the operating table, and likewise no loss of time in starting the appropriate postoperative care. The treatment of the medical emergencies that are bound to arise in these patients should be met with the same promptness as the surgical emergencies, and as these must first be cared for by the resident staff it is obviously advantageous that the internes who cared for the patient before operation should care for him afterward. The strictly medical complications, however, should be observed by the medical resident who has at his disposal the appropriate laboratory facilities as well as the training which allows him to carry out a program based on the principles laid down by the internist. The organization should be so regulated that the internist can easily keep in close touch with the progress of the patient during the operative period.

Divided or indefinite responsibility, on the other hand, brings unsatisfactory results;

therefore, the surgeon must assume the final responsibility. That he and his assistants should avail themselves of the benefits of the internist and the latter's assistants and laboratory facilities, is only a matter of common sense; the exact method will vary under different circumstances, but all methods will revolve around the essential point which, in brief, is maintaining unbroken the continuity of the pre-operative and postoperative treatment, and the prompt meeting of medical as well as surgical emergencies; the former, when promptly and correctly handled, can often be prevented from becoming serious.

The surgical mortality rate in cases of exophthalmic goiter and of diabetes in which operation must be performed is reduced to a certain minimum by strictly surgical technique, skill, and judgment, and a material reduction of this minimum should be possible by the co-operation of the internist and surgeon, and their respective staffs.

That there has been a steady decrease in the mortality rate following surgery in cases of diabetes is shown by the statistics in Joslin's monograph. The following figures quoted in part illustrate the general results obtained. At the Massachusetts General Hospital preceding 1918, Fitz reported a mortality of 30 per cent; between 1918 and 1922, Young found that it was reduced to 15 per cent. Strouse, at the Michael Reese Hospital in Chicago in 1916, reported the mortality rate as 31 per cent, although there were no deaths among eight patients who were properly prepared. Karewski, at Berlin, in 1914, reported a mortality rate of 12 per cent after operations on aseptic tissue, and of 22 per cent after operations on infected tissues. Berkman, at the Mayo Clinic in 1915, reported a mortality of 8 per cent in 26 operations, and in 1921 of 6.4 per cent in 233 operations, which at that time was a remarkable improvement. In Joslin's own cases the rate, up to January, 1917, was 18 per cent, and since April 1, 1919, it has fallen to 9 per cent in 61 operations. A further step in advance has been made during the last two years by Wilder and Adams, who report from the Mayo Clinic that as a result of an appropriate dietary control, and the proper administration of insulin, the mortality rate has been reduced in a series of 327 operations, including those for gangrene, on 251 diabetic patients to 1.2 per cent by operation, and 1.6 per cent by case; 141 of these operations were major surgical procedures, among which were 83 intraperitoneal operations with a mortality of 3.6 per cent. There were also 26 thyroidectomies, 5 nephrectomies, and 7 thigh operations in cases of gangrene, without a fatality.

Similarly the mortality rate in exophthalmic goiter has been reduced from the high figures that occurred in the past to 1 per cent by operation and 1.7 per cent by case, as recently reported by Pemberton. A great part of this reduction has been due to the discovery by Plummer that the peculiar and characteristic crises of this disease can be eliminated by the administration of iodine and that also this drug will prevent, in large part, the postoperative hyperthyroid resection which so often results in death. Those of us who have seen the benefits in diabetic surgery from the proper administration of insulin and appropriate dietary control are impressed with the similarity of the results obtained by the use of iodine and proper diet in cases of exophthalmic goiter.

As a result of co-operation between the surgeon and internist, the surgical mortality in exophthalmic goiter and in diabetes may be compared favorably with the death rate from similar major surgical operations on patients in whom the risk of operation is not increased by a serious and intrinsically dangerous metabolic disease. WALTER M. BOOTHBY.

MASTER SURGEONS OF AMERICA

MOSES GUNN

DR. GUNN was born in East Bloomfield, Ontario County, New York, April 20, 1822, and was the youngest of four children. He died in Chicago, November 4, 1887. His father, Linus Gunn, a prosperous and well-to-do farmer, and his mother, Esther (Bronson) Gunn, were born in Massachusetts, of Scotch Laird ancestry. They were of strong character, efficient, and were Protestant Christians.

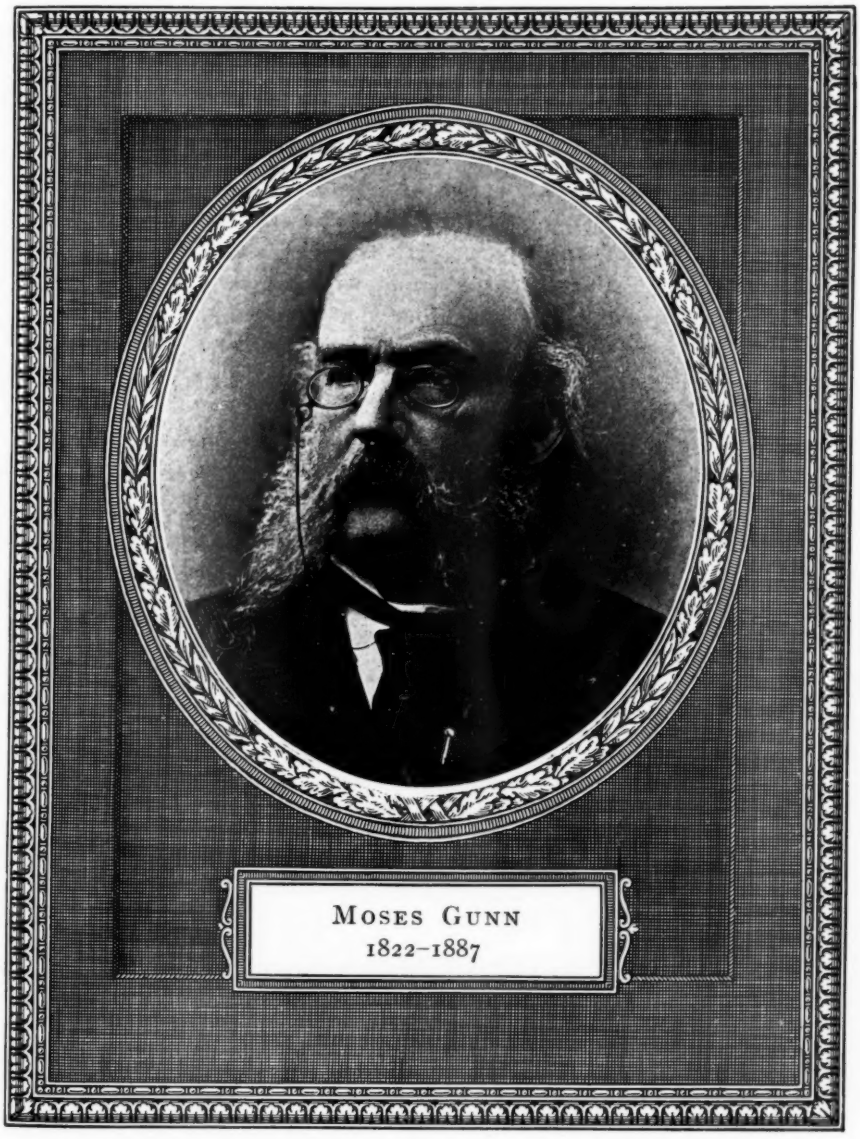
At an early age Dr. Gunn attended the schools of his neighborhood. At the age of 12 he was placed under a tutor, a theological student, who continued to teach him for three years. After that he entered the Bloomfield Academy which he attended until he became ill with pleurisy and empyema, which made him an invalid for several years. He told me the story of this early illness with elaborate details when he was quite sixty years old. After a long time, he said, the pus "pointed" in the side, broke through a minute opening—surprised him one day by wetting his side—where it thereafter drained into dressings for many months. It finally healed but his side was sunken and his shoulder dropped. Although he lacked productive cough, he was said at one time to have consumption and to be slowly approaching death. In spite of this he spent years with hard work and laborious exercises to straighten his body, which he finally succeeded in doing, and when I first saw him at Ann Arbor in 1866 he was a perfect Apollo in appearance, and so continued until his final sickness.

He must have begun the study of medicine about 1842, and was doubtless moved to do so by his precarious health. He had a preceptor in Dr. Edson Carr, of Canandaigua, New York.

In 1844 he entered the Medical Institution of Geneva, New York[1]; and was graduated in 1846 after two courses of lectures of seven months each. During his second year he assisted the demonstrator of anatomy, Dr. Corydon L. Ford, acquiring a great fancy for practical anatomy.

He was prevented from going to college by his protracted sickness; but he was a universal student through life. He received two honorary degrees: the master of arts from Geneva College in 1856, and the doctor of laws from the Chicago University in 1877.

[1] In 1861 this school became the "Geneva Medical College." It was closed finally in 1872. Later it became the Medical Department of the Syracuse University.



MOSES GUNN
1822–1887

His enthusiasm for anatomy and surgery and his personal ambition and personal force were such that in a week after his graduation he started, in February, 1846, for Ann Arbor, Michigan, and at once began a private course of lectures on anatomy to two dozen students of the young state university and a few medical practitioners. He had brought with him from Geneva a huge cadaver. These were the first lectures of the sort in the State of Michigan.

He did some practice and continued private lectures on anatomy and surgery until 1849, when the nascent medical department of the university needed a professor of anatomy. He had already developed such a reputation for scholarship and teaching that over strong competition he won the place. In January, 1850, the chair of surgery was founded, and he was appointed to that likewise. In the winter of 1849 and 1850, before beginning his lectures on surgery he made inspection visits to the medical schools and hospitals of New York, Boston, and Philadelphia. His first class in surgery at Ann Arbor numbered 92; his last class (in 1866–1867) 525. In 1854 he resigned the chair of anatomy to Dr. Ford, of wide fame as a teacher of anatomy for a quarter of a century at Ann Arbor and elsewhere, but he continued in surgery until he went to Chicago, in 1867.

He was married to Miss Jane Ferry in 1848. He moved his residence to Detroit in 1853, but journeyed to Ann Arbor twice a week thereafter during term time to lecture. In Detroit he engaged in general practice for a number of years, to confine himself to surgery later.

He was a regimental surgeon in the Army of the Potomac in 1861–1862, and was at the battle of Williamsburg. General McClellan was his hero, in whom he could see no fault. His partizanship was shown in numerous letters to his wife, which she published in a worthy book of memoirs of Dr. Gunn, after his death. His army service was severe on his health, and he returned a thin and debilitated man.

He went to Europe with doctor friends in 1879 better to recuperate from an attack of septicæmia from an arm infection. Accompanied by his wife he visited Europe again in 1881 for a pleasure trip.

In 1867 he resigned from the University of Michigan and accepted the professorship of surgery in Rush Medical College, where he remained until his death. He gave here twenty years of brilliant service in didactic and clinical surgery. He was a teacher born—his lectures were brilliant and would have made good literature if printed exactly as uttered.

He was a man of striking personality and character. Tall, erect, straight, and well proportioned, graceful in movement, fastidious in taste and action—and in clothes—it was all accentuated by his Burnside beard and long hair, made into ample ringlets each morning by the insistent fingers of his devoted wife. He frequently rode horseback to his lectures; and was a striking figure mounted on the handsomest horse procurable. He never boasted, and was not vain, although

some who did not know him guessed that he was. He was a consistent churchman (Episcopalian), a cheerful Christian, and something of an optimist.

He had many of the best traits. He was meticulously truthful and exact in his words, and absolutely clean in speech. He was fair to others, true to his character and profession, and too full of the business of life to indulge in jealousy or the disparagement of others.

He was a man of great industry and study. He was a fine general scholar—a speaking German scholar, and a fair French one. He had much joy in amateur astronomy, and had a telescope mounted in his house. One of his cardinal virtues was that of punctuality in all appointments. No doctor ever charged him with tardiness at a consultation.

He was an elegant operator, and in every way a superb surgeon. He studied his cases, and his work was singularly free from unexpected incidents, and he had no ornamental gestures or unnecessary talk or actions at his operations. To him the body of his patient was sacred, and an operation was a serious business.

His one or two prolonged clinics each week at the College meant a great surgical service. He operated regularly at the Cook County Hospital and the Presbyterian Hospital, and occasionally at other hospitals in Chicago.

For a man of his age in the profession when aseptic surgery came into vogue, he adjusted himself to it with surprising facility and faith—and great satisfaction.

On occasion at Detroit he was an editor of medical journals. His writings and occasional addresses were scholarly utterances without verbiage or excessive statements. His lectures were all exercises in general culture.

His teachings always ennobled medicine and surgery; they were never allowed to lag in dignity or intense interest to his classes. He made many minor and one great contribution to the science of surgery. This was his work on dislocations of the hip and shoulder joints. The reduction of these dislocations had always been to surgeons a melancholy source of great labor, failures, and awful, sometimes brutal, treatments by great force. By laborious and painstaking work in dissections and manipulations, he showed that by putting the bone in the exact position that it had at the moment of dislocation and then exercising moderate reverse force the bone passed into normal position easily, without great strain or suffering. This doctrine is so logical and self-evident, and has proved so true and satisfactory in actual practice that it has stood substantially without challenge. He announced his theory before the Detroit Medical Society in 1853, thus early in his career. He reaffirmed it in 1859 with amplifications; and he restated it with ampler illustrations in 1884, only three years before his death.[1]

NORMAN BRIDGE.

[1] Chicago M. J. & Examiner, 1884, No. 5.

TRANSACTIONS OF SOCIETIES

CHICAGO SURGICAL SOCIETY

REGULAR MEETING HELD APRIL 6, 1923, DR. FREDERICK G. DYAS, PRESIDING

ADHESIONS ABOUT THE ASCENDING COLON SIMULATING CHRONIC APPENDICITIS

DR. CHARLES DAVISON read a paper on "Adhesions about the Ascending Colon Simulating Chronic Appendicitis." (See page 171.)

DISCUSSION

DR. DON J. ROYER (by invitation): The excellent paper just presented has covered the subject very thoroughly and little remains to be said aside from the roentgenologic aspects. These cases mentioned were diagnosed during careful, routine examinations of the gastro-intestinal tract. In addition to the 10 cases cited, at least 5 or 6 others have been found, two of which were verified at operation. Dr. K. A. Meyer saw one of these latter, in which the adhesions extending from the parietal peritoneum upward and inward across the ascending colon to the transverse colon, were very dense and fibrous, and almost the thickness of a man's hand. Roentgenoscopy and roentgenography are both essential in establishing a positive diagnosis. There is a marked difference in the roentgen findings of these adhesions, and of those of inflammatory origin. A few cases presented evidence of both conditions. It requires no special skill or equipment to diagnose this condition by means of the roentgen ray, but a careful, complete examination of the entire gastro-intestinal tract is of the utmost importance.

DR. WILLIAM M. HARSHA: In using an enema or barium meal in the diagnosis, which do you consider the more important?

DR. DAVISON: The barium meal is the more important. In many of them we use both.

DR. JOHN R. HARGER: Recently two cases have come under my observation—both of which presented a condition from an X-ray standpoint, similar to what Dr. Davison has just presented. In the one case the first part of the transverse colon ran parallel to and just to the medial side of the ascending colon for a distance almost the length of the ascending colon. This patient has had several attacks resembling a low grade appendicitis but has not yet submitted to operation even though a diagnosis of some form of adhesions has been made.

In the other case the first part of the transverse colon ran parallel to the ascending colon and immediately in front of it, then extending just to the right of the cæcum and on into the pelvis. In this case the attacks of pain were referred to the region of the hepatic flexure and upper part of the ascending colon. At operation no adhesions were found but the entire ascending colon and especially the hepatic flexure was found to have a mesentery from 4 to 6 inches long, permitting a very free movement and no doubt at times a kinking of the hepatic flexure. In both cases there was a distinct filling defect at the flexure.

DR. R. W. MCNEALY: I would like to ask Dr. Davison if he considers these membranes or adhesions in any way different from the membranes described by Jackson. The old Jackson membrane picture was considered a congenital affair, and he states that one-half of these cases have been operated on while others have not been operated on, although showing practically the same pathology. Whether they are considered adhesions at the primary operation, or whether they are all congenital adhesions is a question, and if the latter, do they differ from the membranes described by Jackson? Are they the same thing, or is this a report of the finding of Jackson's membrane? It looks as though it extends from the parietal peritoneum over the ascending colon and attaches itself to parts of the transverse colon.

DR. DAVISON (closing): These patients complain of errors of digestion continuing for some time after the ingestion of food, suggesting duodenal ulcer. They constantly complain of moving gas in the intestines. The cases reported were X-rayed, in different positions, from the œsophagus down to the rectum to exclude other lesions. In each case the diagnosis was definitely established by X-ray and the diagnosis confirmed at operation.

This lesion is entirely different from the membrane described by Jackson. Jackson's veil extends over the lower part of the ascending colon. It is so thin that the blood vessels of the colon can be seen through it as it slides back and forth over the intestine. When the intestine is freed from Jackson's membrane the colon assumes its proper size, shape, and position at once, and the peritoneum of the colon beneath this membrane is not abraded.

In the adhesions which we have described these conditions are different. There is actual attachment between the colon and the adhesion and when they are separated at the line of cleavage, leaving a raw

surface, a peritoneal layer is not apparent. The adhesion is, in all probability, due to a long continued, low grade infection from the colon, with deposition of new tissue. As the tissue organizes and contracts it decreases the size of the colon at that point and rotates the colon in the direction of the fixed attachment of the adhesion. In this manner the stasis of the colon is progressive.

Regular Meeting Held May 4, 1923, Dr. Frederick G. Dyas, Presiding

THE ISOLATION OF THE SUBMUCOSA

Dr. A. J. Graham read a paper on "The isolation of the Submucosa as an Aid in Intestinal Anastomosis." (See page 259.)

DISCUSSION

Dr. A. J. Ochsner: This scholarly paper of Dr. Graham's deserves careful consideration because it represents an enormous amount of careful work, and it is the first paper since the one written by Dr. Halsted which emphasizes the importance of the submucous connective tissue layer of the alimentary tube as regards its importance in intestinal surgery.

Since the remarkable work by Lembert, over one hundred years ago, published, I think, in 1812, which described the first real successful method of intestinal suture, every successful method which has been described was successful because it utilized the submucous connective tissue layer; and the success depended entirely upon the proper use of this tissue. But sufficient stress has not been laid upon this fact, and for this reason Dr. Graham's paper should be thoroughly appreciated.

Dr. Alfred A. Strauss: I think that Dr. Graham has called the attention of the profession to a very important and very vital point in gastro-intestinal surgery, namely, the importance that the submucosa plays in forming a union that is safe from leakage.

I think the practical point of application of this interesting work that Dr. Graham has done is that when the mucosa and submucosa are sutured as one layer the muscular layer and peritoneum as another layer, the inner suture, which is composed of mucosa and submucosa, constitutes the vital parts of the strength of the bowel. There is also an exudate and serum poured out between the inner layer and outer layer of sutures which acts as a double safeguard as to leakage. Suturing these layers distinctly does not cut off the important blood supply that is present between the submucosa and muscularis, while in those types of sutures where the suture dips down through all the layers to the submucosa, there is bound to be a strangulation of the blood supply and more of an anæmic necrosis which cuts through and is more apt to produce leakage.

I have demonstrated from animal experiments that the best suture for getting perfect approximation and a good union without any scar formation is a simple over and over suture, the inner one catching the mucosa and submucosa slightly on the slant so as to catch a large surface of the submucosa and practically holds the mucosa edge to edge. The importance of this stitch is that it is the strongest layer of all the coats. The seromuscular suture can also be sutured edge to edge just as you close the skin by an over and over suture, in the type of a Lembert suture, which simply inverts the muscularis very slightly.

Dr. Graham (closing): In a resection (amputation) of a portion of bowel, the major injury to the organism, and the one to which expression is given immediately, is that to the circulation. The blood is thrown against the closed ends of the arteries, the serum exudes and clots, forming the plastic exudate. Lembert took advantage of this *immediate* exudate in approximating the peritoneal coats to which it would adhere. With the sealing power of this exudate any stitch, securing the submucosa and firm approximation of the peritoneum, will obtain a tight joint.

Mit Keyſerlicher freyheit getruckt zů Straßburg durch Joannē Schott.

THE SURGEON'S LIBRARY

OLD MASTERPIECES IN SURGERY

By ALFRED J. BROWN, M.D., F.A.C.S., Omaha

THE FIELD BOOK OF THE TREATMENT OF WOUNDS

THE latter part of the fifteenth and beginning of the sixteenth centuries saw much more attention paid to the care and treatment of wounds; and surgeons whose province it was to care for the wounded, were attached to the armies in the field. Jerome of Brunswick had written his surgery in 1497 and thus opened the way for amplification of methods. In 1517 Hans von Gerssdorff called Schylhans who describes himself as a citizen and wound treater of Strassburg, published his *Feldbuch der Wundartzney* which was printed in Strassburg by Joann Schott. It is illustrated by many woodcuts attributed to Hans Pilgrim known as Waechtelin. Some of these are original while others seem to be more careful drawings and better prints of the illustrations of Jerome's surgery.

The *Feldbuch* contains the first known illustration of an amputation. The patient is seated in a chair. The leg which is being amputated is held by an assistant. A cord is wound tightly around the leg above and another below the site of amputation but the blood is shown pouring in streams from two holes in the proximal stump. A patient whose left hand is bandaged stands calmly watching the procedure while the surgeon wields the saw. The legends to the illustrations are all in poetry and many of them are extremely complimentary to Gerssdorff who apparently has no intention of hiding his light under a bushel. The legend over the amputation illustration reads:

"Arm bein abschniden hat sein kunst
Vertriben sanct Antonien brunst.
Gehort auch nit eim yeden zu
Er schick sich dan wie ich im thu."

To cure Saint Anthony's fiery smart
Removing arms has certain art
Which is not in all men 'tis true
So send your case to me to do.

To be sure Gerssdorff has a good excuse for this self esteem, for he states he had performed between one and two hundred amputations, probably many more than any other surgeon up to that time. He says that he has heard that giving patients drinks to make them sleep makes them delirious. So in his amputations, he has never given drinks, but describes a method of using opium, gradually given, which causes sleep. He then wakes his patient by the inhalation of vinegar. He does not ligate vessels in the stump but uses the cautery or caustic plaster to check the bleeding. The stump is enclosed in the bladder of a bull, ox, or hog.

Another interesting illustration is that of the wounded man, a later evolutionary type of the old zodiacal diagrams, which shows different wounds and the various implements which cause them. These wounds are placed at the proper positions for bloodletting or ligation of vessels. The legend over the illustration reads:

"Wiewol ich bin voll Streich un(d) Stich
Zermorsscht verwundet iamerlich
Doch hoff ich Gott kunstlich Artzney
Schylhans der werd mir helfen frey."

When I am stricken hip and thigh
Or wounded greviously do lie
I hope that God will bring to me
Schylhans' artistic surgery.

Gerssdorff places great stress on anatomy. This portion of the book is illustrated by a plate of the opened torso attributed to Wendelin Hock and probably engraved by Waechtelin called Pilgrim and a skeleton which is commonly known as the Waechtelin skeleton. In the surgical portion of the work he emphasizes constantly the fact that the surgeon does his work with his hands. The very definition of the word shows this as he says: The name Chirurgicus springs from *chir* in the greek tongue which means a hand and *gios*, using; hence a surgeon is a hand worker or wound physician.

Among other things Schylhans made and illustrated a trivalve speculum for use in the vulva and anus which is an improvement over the bivalve speculum illustrated by Jerome of Brunswick.

In addition to the classification of wounds and fractures and their treatment he classifies the various medicaments used according to their action and defines such action as styptics, sedatives, etc. He adds a materia medica and the proper dosage.

Gerssdorff takes up other common diseases in addition to wounds. He discusses fistula and carbuncle. The pestilence he calls anthrax. Cancer and leprosy he considers incurable but gives methods of treatment which he calls palliative. Ergotism he describes as "ignis sacer" or St. Anthony's fire and pictures a sufferer from it holding up a gangrenous hand which has burst into flame to St. Anthony and appealing to him for relief. For the cure of severe cases Gerssdorff advises amputation.

REVIEWS OF NEW BOOKS IN SURGERY

I suppose that the average individual, either medical or lay, would be inclined to doubt seriously whether there is such a thing as romance in the bacterial world, that is if he ever gave the matter a thought. Yet in this book[1], a cold blooded scientist, one who is thoroughly familiar with the ways and doings of the microscopic world of bacteria, brings out the romance in that world so that it reads like a best seller. It is not a textbook, yet it was written to instruct and to make the instruction palatable. And in that it has succeeded. Neither is it a kindergarten book written in words of one syllable. There is meat in it that will test the digestion of the wisest medical man.

It can be recommended as an antidote for much of the present day "microbiphobia," and for that alone it is a good book for the physician to place in the hands of his patients. Furthermore the physician himself can find food for thought in the chapter on "Immunity, Susceptibility and Resistance to Infection." In this there is a distinctly new angle.

The book has another merit; it is a synthesis, that is the author brings knowledge from many different angles to bear on the subject, and out of this he builds up the broad general principles that underlie the world of bacteria. It is an unusual book.

B. H. MOORE

ONE welcomes the appearance of a new edition of this most excellent book.[2] Of especial interest is the publication in one volume instead of two as was the case with the first edition. None of the colored plates of the latter have been omitted and much new material both in the way of text and illustrations added. The chapter on optical principles and the interpretation of images has been rewritten so as to make it less technical.

It is not easy for those accusomed to working with cystoscopes giving images which have been corrected by lenses so as to be non-inverted to become accustomed to many of the colored plates in this book, and it is to be hoped that for American readers at least the inverted images will be omitted in future. The authors suggest the use of an apparatus to accomplish this but inasmuch as most German, English, and our own books on cystoscopy have made these changes we fail to see the necessity of making these corrections from inverted to non-inverted views by the employment of a special apparatus.

Another suggestion for future editions is that an English or American urologist should be called upon to write the English legends. The use of the term "swelling" for ureteral papilla or such a legend as "swelling of bladder by retroverse uterus" or the employment of "bladder ground" for floor or "foreside" for anterior wall are only a few of many regrettable errors, especially in view of the fact that the German and Italian legends are uniformly correctly translated.

The outstanding features of the book are its colored plates, and even if one could not read a word of the text, a glance from time to time at this most remarkable collection of cystoscopic views would well repay the purchase.

The perusal of the chapter on posterior urethroscopy reveals the fact that French urologists have adopted the McCarthy instrument in preference to any European one. The chapter on pyelography has been greatly enlarged in this edition but considering the vast material at the disposal of the authors many more diseases should be included than is the case. One misses reference to our methods of gradual dilatation of the ureter by employing a number of bougies, in the section on non-operative delivery of ureteral calculi. The subject of the use of the high frequency current is very thoroughly covered.

The book is more than a simple treatise on cystoscopy and ureteral catheterization, and really includes many subjects which are not ordinarily taken up in our monographs, hence its appearance in an English edition is very desirable.

DANIEL N. EISENDRATH

[1] CIVILIZATION AND THE MICROBE. By Arthur I. Kendall. New York: Houghton Mifflin Co., 1923.

[2] TRAITÉ PRATIQUE DE CYSTOSCOPIE ET DE CATHÉTÉRISME URÉTÉRAL. By G. Marion and M. Heitz-Boyer. 2d ed. Paris: Masson et Cie, 1923.

BOOKS RECEIVED

Books received are acknowledged in this department, and such acknowledgement must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

AERZTLICHE BEHELFSTECHNIK. Bearbeitet von C. Franz, Berlin; Th. Fuerst, Muenchen; R. Hesse, Graz; K. Holtei, Gratwein; H. Huebner, Elberfeld; O. Mayer, Wien; B. Mayrhofer, Innsbruck; G. von Saar, Innsbruck; H. Spitzy, Wien; M. Stolz, Graz; R. von den Velden, Berlin; Herausgegeben von Professor Dr. G. Freiherr von Saar. Bearbeitet von Professor Dr. Carl Franz. 2d ed. Berlin: Julius Springer, 1923.

MENTAL HYGIENE AND THE PUBLIC HEALTH NURSE; PRACTICAL SUGGESTIONS FOR THE NURSE OF TODAY. By V. May MacDonald, R.N. With a foreword by Thomas W. Salmon, M.D. Philadelphia and London: J. B. Lippincott Co., 1923.

RUBBER AND GUTTA PERCHA INJECTIONS; FOR RAISING THE DEPRESSED NASAL BRIDGE AND ALTERING EXTERNAL CONTOURS. By Charles Conrad Miller, M.D. Chicago: Oak Printing & Publishing Co., 1923.

THE AMERICAN ILLUSTRATED MEDICAL DICTIONARY. A new and complete dictionary of the terms used in Medicine, Surgery, Dentistry, Pharmacy, Chemistry, Nursing, Veterinary Science, Biology, Medical Biography, etc., with the Pronunciation, Derivation, and Definition. 12th ed., revised and enlarged. By W. A. Newman Dorland, A.M. M.D., F.A.C.S. Philadelphia and London: W. B. Saunders Company, 1923.

PEDIATRICS. By Various Authors. Edited by Isaac A. Abt, M.D. vols. i. and ii. Philadelphia and London: W. B. Saunders Company, 1923.

THE EXAMINATION OF PATIENTS. By Nellis B. Foster, M.D. Philadelphia and London: W. B. Saunders Company, 1923.

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LES ULCÈRES DE L'ESTOMAC ET DU DUODÉNUM. By Ed. Enriquez et Gaston-Durand. Paris: Masson et Cie, 1924.

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ESSENTIALS OF ORAL SURGERY. By Vilray Papin Blair, A.M., M.D., F.A.C.S., and Robert Henry Ivy, M.D., D.D.S., F.A.C.S. St. Louis: C. V. Mosby Company, 1923.

LE SYMPHATIQUE CERVICO-THORACIQUE. By Prof. Thomas Jonnesco. Paris: Masson et Cie, 1923.

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CLEFT LIP AND PALATE. By Truman W. Brophy, M.D., F.A.C.S. Philadelphia: P. Blakiston's Son & Co., 1923.

DIAGNOSIS AND TREATMENT OF ACUTE ABDOMINAL DISEASES, INCLUDING ABDOMINAL INJURIES AND THE COMPLICATIONS OF EXTERNAL HERNIA. 2d ed. By Joseph E. Adams, M.B., M.S., (Lond.), F.R.C.S., (Eng.) New York: William Wood & Co., 1923.

A TREATISE ON ORTHOPEDIC SURGERY. By Royal Whitman, M.D., M.R.C.S., F.A.C.S. 7th ed. rev. Philadelphia and New York: Lea & Febiger, 1923.

GREEN'S MANUAL OF PATHOLOGY AND MORBID ANATOMY. 13th ed. rev. By W. Cecil Bosanquet, M.A., M.D., (Oxon.), F.R.C.P. (Lond.), and G. S. Wilson, M.D., M.R.C.P., D.P.H. (Lond.) Philadelphia and New York: Lea & Febiger, 1923.

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THE MEDICAL DEPARTMENT OF THE UNITED STATES ARMY IN THE WORLD WAR. vol. v.—MILITARY HOSPITALS IN THE UNITED STATES. By Lieut. Col. Frank W. Weed, M.C., U. S. Army. Prepared under the Direction of Maj. Gen. M. W. Ireland, M.D. Washington: Government Printing Office, 1923.

BIOLOGIE UND PATHOLOGIE DES WEIBES. EIN HANDBUCH DER FRAUENHEILKUNDE UND GEBURTSHILFE. Edited by Josef Halban, Wien, and Ludwig Seitz, Frankfurt a.M.. No. 2.—GESCHICHTE DER GYNAEKOLOGIE. By Priv.-Doz. Dr. I. Fischer, Wien; NORMALE ENTWICKLUNGSGESCHICHTE DER WEIBLICHEN GESCHLECHTSORGANE DES MENSCHEN. By Professor Dr. W. Lubosch, Wuerzburg. No. 3.—DIE KONSTITUTIONSTYPEN DES WEIBES, INSBESONDERE DER INTERSEXUELLE TYPUS. By Prof. Dr. P. Mathes, Innsbruck; VEGETATIONS- UND WACHSTUMSSTOERUNGEN, OSTEOMALACIE, CHLOROSE. By Prof. Dr. H. Guggisberg, Bern. Berlin: Urban & Schwarzenburg, 1924.

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AMERICAN COLLEGE OF SURGEONS

THE LAYING OF THE CORNER STONE OF THE JOHN B. MURPHY MEMORIAL BUILDING

THE great desire expressed by the representatives of all classes of Chicago citizens to provide a fitting memorial to John B. Murphy, the man whose scientific achievements have made his name familiar throughout the world, and the results of whose work were so vital in their importance and so original in character that they are more and more appreciated as time passes, led to the formation of a definite plan and an organization for the purpose of making possible its realization.

The form of memorial, fitly chosen by those in charge, was living in character, that is, a great organization devoted to the scientific pursuits to which Murphy gave his life and of which he was one of the founders and a leading and most devoted member; its tangible form being a great building especially designed to meet the requirements of the American College of Surgeons.

The work of securing the required funds occupied two years and at times seemed to be impossible of success, in the main, because men of means had become weary from the many requests upon them for financial assistance in behalf of innumerable projects. The hope was realized through the active and self-sacrificing co-operation of Doctor Murphy's family, friends and admirers, together with the organized effort of a large number of members of the medical profession from the Atlantic to the Pacific Ocean, who were appreciative of his achievements and who also knew that the memorial building will be of direct great value to the work of the organization which was chosen as the recipient.

On October 23, 1923, a very notable event occurred in the history of Chicago in that on that day was the laying of the corner stone of the John B. Murphy Memorial Building with appropriate ceremony. A great throng was present composed of representatives of all classes, and the exercises, although very brief were most impressive in character. Among those who took part were Mr. Leroy A. Goddard, president of the Memorial Association, who presided in place of the Honorable William A. Dever who was unexpectedly called from the city, Dr. William J. Mayo, the daughters of Dr. Murphy, and other distinguished guests resident in the city, leading members of the medical profession, a great number of the Fellows of the College, and the Board of Regents who gave a touch of color due to their caps and gowns of crimson and royal purple. The real significance of this occasion, which undoubtedly was apparent to all, was due to the fact that the beautiful building to be erected will serve as a memorial to the man who was such an honor to his city and country on account of the value of his life's work and also because the memorial will necessarily be looked upon, as it is, as one of the few great evidences of the world's appreciation of the services which have been rendered by the medical profession to suffering humanity.

ADDRESS OF WILLIAM J. MAYO

DR. WILLIAM J. MAYO: The American College of Surgeons was organized in response to a great human need. Modern surgery had developed with such extraordinary rapidity that it had outgrown the existing methods of medical education. Surgery in the pre-antiseptic day was confined largely to necessary life-saving operations in emergency cases. The medical man who first saw the patient had to care for him, and this, of course, is still true in certain acute conditions. In a vital emergency, such as hæmorrhage or acute obstruction of the bowels, it is most important that operation be quickly performed. Early operation, even by one who operates only occasionally, gives better results than those achieved in the later stages by the greatest surgeon in the world. As surgical science advanced, however, many medical men with the best intentions attempted operations which were not imperative, and for which they had not the training, knowledge, or skill. It may be said in extenuation that surgical training was not easy to obtain at this earlier period; the large majority of operators learned by experience, profiting by their mistakes. There was no other way. Today there are ample opportunities for surgical training, yet of 150,000 medical practitioners in the United States, more than 50,000 are performing operations, only about 10,000 of whom are well qualified surgeons.

There is a sufficient number of trained surgeons in America to do the necessary work, and the untrained men, in justice to the patient, should not undertake surgery.

The founding of the American College of Surgeons was primarily in the interest of the patient. Its purpose was to band together the competent men in general surgery and the men in highly developed surgical specialties, of which ophthalmology, for instance, had already attained a distinctive position, recognized both by the medical profession and the laity. An organization of this magnitude was a colossal undertaking, but the vision and extraordinary organizing ability of Franklin H. Martin made possible the founding of the American College of Surgeons ten years ago. Today the directory of the College contains the names and addresses of more than five thousand responsible surgeons in North America and gives at a glance information with regard to reputable men in all parts of the country competent to perform necessary operations.

In the development of the Fellowship, not only the surgical ability but also the moral character of the candidates was taken into consideration. The man of ability without character who trades commercially on the confidence of the patient is the most dangerous member of the profession.

The American College of Surgeons is not an autocracy controlled by a few men. Before a man can be examined by its officers his qualifications and character must be approved by the central committee of his state. The younger surgeon who applies for fellowship serves what might be called a probation period of from six to eight years after graduating in medicine. Not only must he be trained, but he must show that he is capable of applying the art of surgery in the best interest of the patient. Herein the American College of Surgeons differs from those organizations abroad on which it was modeled, in which knowledge of surgical science alone is required.

Among the many functions of the association is that of aiding the surgeon to make scientific progress. SURGERY, GYNECOLOGY, AND OBSTETRICS, the official organ of the College, is, I believe, the greatest surgical journal in the world, and a powerful influence in the education of the surgical profession. The policies of the journal are controlled by the Board of Regents of the College. Clinical surgical meetings of an educational character are held in various parts of the country each year, which are of great value, and act to keep the Fellows in touch with the latest and best in surgical science.

One of the most important accomplishments of the American College of Surgeons is the standardization of hospitals, recently completed for nearly all the one-hundred-bed hospitals of the country. When this work was undertaken, most of the hospitals, except a few in the great centers, had only the most meager records, and were without pathological data. They were, in fact, little more than boarding houses where any physician might take a patient, and with the assistance of those in the hospital, perform operations. The hospital, in a way, gave unfounded confidence to the patient that the operation would be done properly. It sounded well to say that the hospital had an open staff, but in practice it was extremely bad for the patient. Today nearly all hospitals have adequate records and pathological data, and the staffs are limited to those men who have a right to do surgical work. No achievement of the College has been of more far reaching importance to humanity than this.

The organization has been greatly interested also in the education of the nurse, who has become an adept in many technical specialties. Positions as technicians of various sorts are now held by highly trained nurses, which gives the physician an opportunity to do other work. When one considers that the nurse completes her professional training in three years, and is self-supporting at the same time, and that the physician spends from seven to eight years of time after graduation from high school, and about eight thousand dollars in money for his training, the economic value of the higher grade of trained nurse is plainly evident.

The American College of Surgeons is truly American. As might be expected, it enrolls Canadians, people of our own race and tongue, who are joined to us by indissoluble bonds of sympathy. The organization has recently formed an alliance with the surgeons of Mexico and Central and South America; this alliance has developed a union of scientific interests which will do much to promote international peace and harmony. The intellectual intuition and tact and the beautiful dexterity of the Latin peoples have made their surgeons among the greatest in the world. Our desire for a better understanding and a closer scientific relationship with the Latin Americans is fully and cordially reciprocated by these surgeons. Exchange of visits by the surgeons of North and South America is most inspiring, and productive of lasting good.

The American College of Surgeons is looking to the future. In 1914, it was voted to raise, among the Fellows, an endowment of $1,000,000, the

income of which was to be used for scientific purposes. More than $600,000 of the amount has been secured. In 1920, public-spirited lay citizens of Chicago and a group of the Fellows residing in the city, presented to the College the beautiful home where many important activities are carried on. To the east of it and on the same plot of ground are located the headquarters of the official organ of the College, SURGERY, GYNECOLOGY, AND OBSTETRICS, and by the generous provision of the owner, Dr. Martin, this eventually becomes the property of the American College of Surgeons.

We meet here today in the center of this ground to lay the corner stone of a building which has for its purpose the attainment of the highest ideal of the human race, aiding the sick and the suffering. It stands on a favored spot in the heart of the City of Chicago, which lies so near the geographical center of North America. Its magnificent proportions make possible a library, a museum, and meeting rooms for the various purposes of the Fellows of the College, and in time it will become one of the greatest heritages of the surgical profession of America. We dedicate this fitting monument to the greatest surgeon of his day, John B. Murphy, one of the founders of the College, who gave unsparingly of his strength and talents to aid in the establishment of the organization, and whose noble spirit will always sanctify this ground.

DR. FRANKLIN H. MARTIN: It may be of interest to enumerate here the particular reasons why the American College of Surgeons should seek to build a memorial to Doctor John B. Murphy. May I remark on these reasons as we review the items in this sacred box, which is to be placed in the corner stone:

First, a copy of the first issue of SURGERY, GYNECOLOGY AND OBSTETRICS, of which Doctor Murphy was one of the founders and the first chief of the Editorial Staff, and which is now the official journal of the American College of Surgeons.

Second, a copy of the same journal which issued the call (backed enthusiastically by Doctor Murphy) for the first meeting of the Clinical Congress of Surgeons of North America, the organization which was the forerunner of the American College of Surgeons.

Third, the resolution calling for the organization of the American College of Surgeons, introduced at the New York meeting of the Clinical Congress of Surgeons of North America in 1912, seconded by Doctor Murphy, and supported by him in an impassioned speech.

Fourth, a copy of a photograph of the first Board of Regents of the American College of Surgeons, of which Doctor Murphy was one of the most influential members.

Fifth, a copy of the first directory of the American College of Surgeons (1913) and a copy of the directory of today (1923).

Sixth, in addition to these reminders of Doctor Murphy's personal interest in the American College of Surgeons, we are placing in this box articles of particular interest to Doctor Murphy's friends and dear ones, all of which will be a reminder to those who come upon its contents in the future of this day, October 23, 1923, when his daughters, with loving hands, laid them away. Among these articles are family mementos, copies of the Chicago daily papers of this day, and a copy of the remarks on this occasion by Dr. William J. Mayo.

However, Mr. President, this but represents our warm friendship for a great man. The seed that is placed here by twenty-five hundred of the confrères of Doctor Murphy was matured from his great work as a teacher, as a citizen, and as a surgeon. By all of this not one iota will be added to his great fame. When in the future generations the contents of this box are revealed, those who survey them will have been conversant with the fame of our great surgeon; but this will serve to emphasize that we who lived with him—his contemporaries, his friends—appreciated him and sought to demonstrate our love by erecting to his memory this monument.

DR. A. J. OCHSNER: I have the honor of laying this corner stone in memory of a charming friend, a noble citizen, and an enthusiastic organizer of the American College of Surgeons, a constant worker for the development of the College of Surgery, the greatest surgical teacher that this city has ever produced, whose words and works will live in the actions and thoughts and words of thousands of his disciples and their disciples.

The corner stone was raised from the blocks and Dr. Ochsner took the trowel, dipped it in the mortar and spread the mortar over the foundation. He was followed by Mrs. Benedict, Mrs. E. N. Hurley, Jr., Mrs. J. T. Murdock, and finally by Dr. Martin.

FELLOWSHIP IN THE AMERICAN COLLEGE OF SURGEONS

By ALBERT J. OCHSNER, M.D., F.A.C.S., Chicago

IT is important that we have a clear idea of what it means to be a Fellow of the American College of Surgeons. It means primarily that the surgeon who has attained this honorable position has been judged and found qualified in all important respects by those who know him most intimately.

The judgment is passed by a credentials committee selected by ballot by the Fellows of the College for each individual state and by a central committee acting for the two continents of North and South America, who weigh carefully all of the facts brought before them through inquiries directed to fellows whose work brings them in contact with the applicant.

QUALIFICATIONS

Honesty. The first and most important qualification is honesty. If the American College of Surgeons cannot conscientiously vouch for the honesty of a surgeon it has no right to include him in its Fellowship because it would not be just to the public. A dishonest surgeon, no matter how skillful, is a menace to the community.

Ability. The second qualification is ability. A man without ability should not be vouched for to the public which has no means of determining whether the surgeon possesses the ability to diagnose the disease or the necessary skill to perform the serious work which he may have to undertake. An honest man with less ability is far safer than a dishonest man with greater ability. But the College must demand both honesty and ability.

Education. There was a time when educational facilities were so meager in this country that many most excellent surgeons were produced through almost superhuman effort although greatly handicapped through lack of educational opportunities, but in every case these men made use of all the available opportunities. Consequently, the College of Surgeons has a right to demand that the present generation make use of the improved conditions which are now available. It is proper that, at least for all of the younger applicants, a very high degree of educational qualification be demanded.

Experience. The older surgeons can remember when only few opportunities for gaining experience were available before entering individual practice. The hospital positions for young men were few; there were no fellowships and very few assistantships, consequently the young surgeon was compelled to accumulate his experience in his personal practice.

At the present time many such positions are available and consequently it is right that the young surgeon shall accumulate a large experience in the service as an assistant of an older experienced surgeon before taking the responsibility of independent surgical work of a serious character in which his inexperience might endanger the life or health of the patient. It is, therefore, right that the College should require experience as one of the qualifications for Fellowship.

Human interest. No surgeon can become a useful member of society unless he shows human interest in those who entrust themselves to his care.

A selfish surgeon lacking human interest is a menace to a community and does not deserve the prestige given him by Fellowship in the College.

Industry. Unless a surgeon is industrious he will soon become inefficient, because he does not keep up with the advances of the surgical profession. Such a member of the profession has no right to enjoy the benefits of Fellowship. His example is especially harmful for the development of the younger generation of surgeons.

Ethics. In order to build up the surgical profession of a community, it is important that the ethical relations toward other surgeons and to the public be correct.

There is a close relationship between the first and last requirements. A surgeon who is absolutely honest can not well be unethical and vice versa; but there are certain relations, in which custom plays an important part, which introduces a difference.

Ethical conduct implies honesty, fair play, and consideration for the other person's interests. It represents the practical application of the Golden Rule both to colleague and client which is often not an easy matter especially in a profession in which the element of competition is so important a part as in the practice of surgery.

There was a time when many of the most honorable members of the surgical profession felt that in order to deal fairly with the practitioner who referred patients to them, a portion of the fee should be paid to the practitioner. The result of this, however, proved most pernicious, because it developed a form of barter in human life. In

many instances the practitioner referred the patient not to the most competent surgeon but to the one who was willing to pay the highest percentage of the fee collected, without regard for his learning, experience, skill, and judgment. This really meant nothing more nor less than barter in human life.

The American College of Surgeons demands of applicants for Fellowship that they be not guilty of this practice and will not be in the future. It makes the practice of splitting of fees in any form an absolute reason for preventing the admission to the American College of Surgeons on the one hand, and a cause for expulsion from the College on the other.

In the large as well as in small communities it is true to human nature that professional jealousies should exist, but the college has a right to expect its Fellows to put aside all personal differences and to support for Fellowship every colleague who possesses the required qualifications without regard to personal likes or dislikes. It would be quite as reprehensible to recommend for Fellowship a friend lacking the proper qualifications as it would be to oppose a candidate possessing these qualifications because of a personal dislike.

It is the duty of every Fellow to encourage young surgeons in the acquisition of the necessary qualifications, because it is exceedingly important to this country to give proper development to the next generation of surgeons.

Selfishness has no place in the acceptance or rejection of candidates for Fellowship.

HISTORIES

The College demands from each applicant that he submit a sufficient number of histories of important cases treated to convince the examining committee; that the applicant make it his practice to give each patient that comes under his treatment a careful physical examination; that he give proper consideration to the clinical history; that he make the necessary laboratory examinations; that he invite consultation when necessary; that he plan the necessary treatment or operations carefully and avoid unnecessary operations; that he show proper surgical judgment and skill in performing the operation; that he record postoperative conditions and end-results.

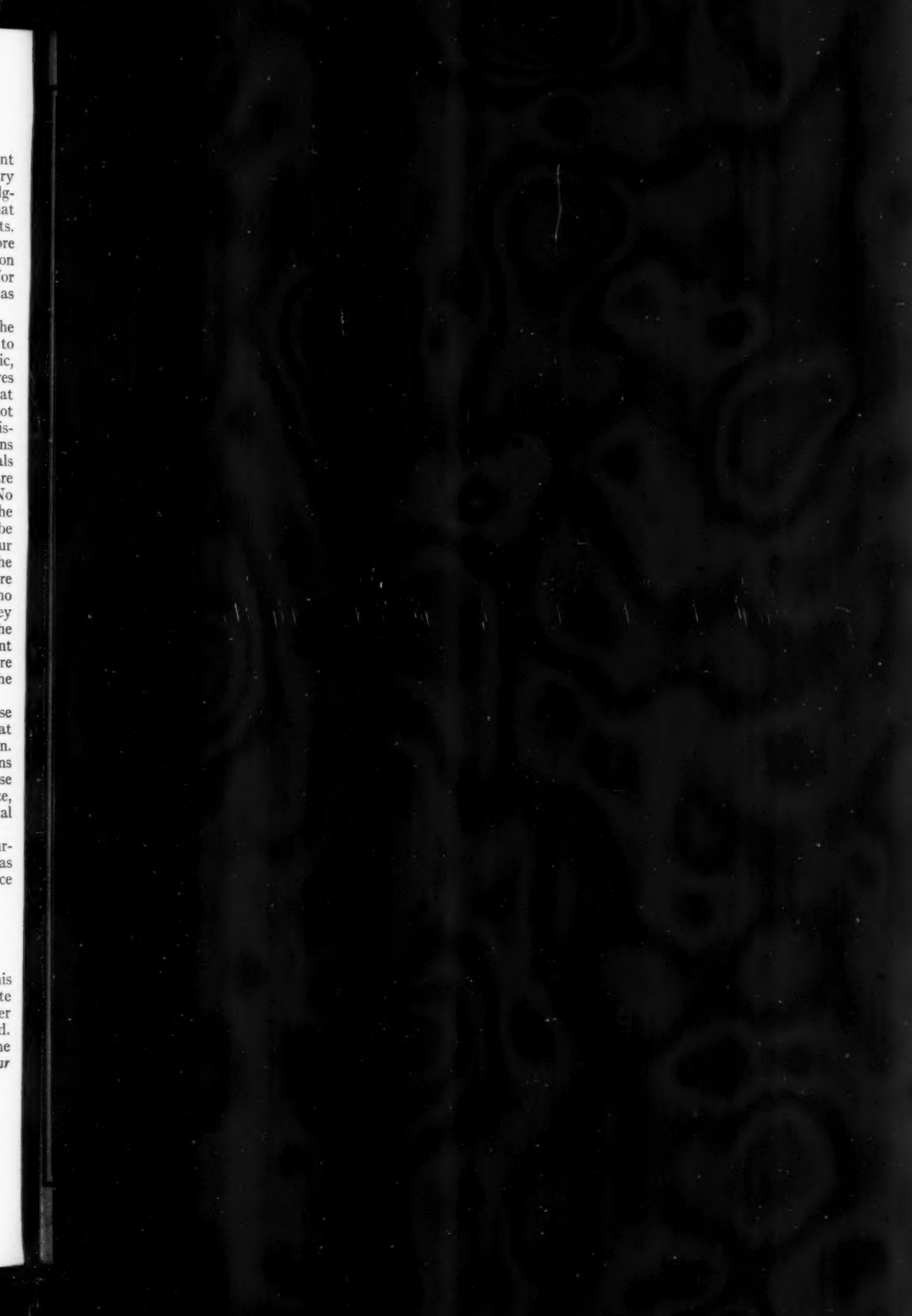
It seems that there can be no method more favorable than this to determine a basis upon which the College can base its judgment for vouching to the public that the surgeon has proper scientific and technical qualifications.

It must be remembered that carelessness on the part of the College in admitting surgeons to Fellowship might cause great harm to the public, because the public has learned to trust their lives in the hands of the Fellows of the College. That a surgeon is not a Fellow of the College does not necessarily mean that he is incompetent or dishonest. There are nearly two thousand surgeons who have applied for Fellowship whose credentials have not convinced the committees that they are competent for admission to Fellowship. No doubt in many of these cases the defects in the records will be cleared up and they will later be admitted, but before they can be admitted your committees must be convinced that all of the requirements have been fulfilled. There are many young surgeons with splendid training who are looking forward to Fellowship as soon as they have acquired the necessary experience. The fact that these are not Fellows at the present time is no discredit to them so long as they are striving in an honorable way to acquire the necessary qualifications.

It is the duty of the Fellows to inspire these young men with the ideals of the College so that they may in due time strengthen the organization.

To the public the American College of Surgeons must mean that its Fellows can be trusted because of their honesty, their learning, their experience, their skill, their human interest, and their ethical appreciation of the Golden Rule.

Fellowship in the American College of Surgeons must mean that every surgeon who has this distinction deserves the absolute confidence of the community in which he lives.

THE COMMITTEE ON THE TREATMENT OF FRACTURES

It is the desire of the Committee on the Treatment of Fractures of the American College of Surgeons that any Fellows of the College who may have constructive ideas concerning the work of this committee or who have been doing special work on fractures should communicate with the chairman, Dr. Charles L. Scudder, 144 Commonwealth Avenue, Boston, Massachusetts. This committee is particularly anxious to have definite ideas and suggestions presented to them in order that the work may be more rapidly developed. (For detailed statement regarding the work of the committee, see pp. 30 and 31 of the *1924 Year Book* of the American College of Surgeons.)